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Original Communications

EXPERIENCES WITH EXTENDING THE INDICATIONS FOR THE USE OF TRACHEAL AND BRONCHIAL GRAFTS

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AN EXTENSIVE literature has already accrued relative to reconstructive surgery of the tracheobronchial tree. Daniel[1] initiated interest and enthusiasm in this field with his presentation before this society during the St. Louis meeting in 1947. He repeated and confirmed the initial experiments done by Taffel[2] in 1940, utilizing free grafts of fascia to cover windows created through the full thickness of the wall of the trachea. He then extended his investigation to include a study of the effect of spanning large gaps in the continuity of the tracheobronchial tree with various types of prostheses. Subsequent experience[3-5] with applications of this type of procedure showed an almost constant tendency of the human trachea to become stenosed severely upon removal of the prosthesis. At the 1949 New Orleans meeting, Gebauer[6] described four highly successful instances of reconstruction of tuberculous bronchial stenosis with full thickness dermal bronchial grafts. In October, 1949, Robb and Bateman[7] described the use of tantalum gauze covered with fascia to effect a repair of the cervical trachea. A successful repair of a traumatic stricture of the bronchus, by excision and end-to-end anastomosis, was reported by Griffith[8] in 1949. The application of resectional procedures with end-to-end anastomosis of the trachea or bronchus was described in 1950 by Maisel and Dingwall[9] and by Ferguson and Wild.[10] In 1951, Mathey and Oustrieres[11] reported successful end-to-end anastomosis in the human after accidental division of the stem bronchus; also in 1951, Paulson[12] described the use of a dermal graft to correct a stricture of the bronchus resultant to severe chest trauma. Methods of tracheal wall reconstruction were reported by one of us (O.A.A.) at the 1948 Quebec meeting of this Association.[13] Quite large defects in the tracheobronchial wall were produced following total removal of the right lung for extensive right upper lobe carcinoma with concomitant resection of the carina, tracheal wall, and

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contralateral bronchial wall. With smaller defects, a transverse closure of the longitudinal ovoid defect constituted a satisfactory reconstruction without decrease in the lumen of the airway. In one patient, a very extensive defect was spanned by use of a prosthesis over which was placed a large pedicle pleural graft. In five instances, operations were performed for extensive carcinoma and, in one instance, for a bronchial adenoma which had extended across the carina down into the contralateral bronchus. This latter patient, who was reconstructed by suture without prosthesis or graft, lived almost four years following surgery free of any pulmonary symptomatology. She succumbed to an extensive neoplastic process in the liver, the exact nature of which could not be determined because of refusal of permission for post-mortem examination. The five patients with advanced carcinoma lived from two months to one year, all succumbing to either local or distal recurrence of the process. The repair of the tracheobronchial ovoid defect by transverse closure necessarily limited the amount of resection of tracheobronchial tissue adjacent to the neoplastic process. In addition, we experienced major difficulty during the postoperative period with the tendency for the airway to become partially or totally obstructed with any shift of the mediastinum. The patient in whom a glass prosthesis was placed because of the need to make a very extensive defect is being re-presented in this publication and will be discussed further in detail.

The initial use of dermal grafts by Gebauer was confined to the treatment of tracheobronchial lesions of the tuberculous type. In 1950, he[14] wrote, " . . . perhaps in time dermal grafts will be used more frequently for nontuberculous tracheobronchial lesions than for the tuberculous type." This prophecy has been partially fulfilled with the reports of the use of grafts in the management of the effects of both acute and posttraumatic stricture. Occasional instances of the use of the procedure for the treatment of intrabronchial tumors have been described in 1947 by Goldman[15] and in 1948 by Langston and Fox.[16] We wish to report our experiences with the utilization of grafting procedures in the management of nontuberculous lesions.

At the present time we feel that there are several indications for considering the application of grafts upon the trachea or bronchus. We submit an outline of these indications for consideration in Table I. There are 3 basic categories: (1) acute trauma; (2) various types of bronchostenosis, both tuberculous and nontuberculous; (3) in the management of various types of different neoplastic processes. There may later be added (4) congenital defects, such as partial agenesis of cartilage. Gebauer[6, 14, 16] and Paulson[12] have emphasized that, in some instances, strictures need not be associated with suppurative destruction of the pulmonary tissue distal to the stricture. In such instances, a repair of the stricture constitutes the only reasonable physiologic surgical approach. Such a situation occurred in Paulson's management of a posttraumatic stricture in which the injury was produced by severe external compressive force upon the thorax. We are reporting herein a similar situation resultant to the intrabronchial trauma incident to the long-standing presence of a metallic foreign body; we assume,

TABLE I. THE PRESENT INDICATIONS FOR CONSIDERATION OF THE POSSIBLE NEED FOR GEBAUER TYPE DERMAL GRAFT

1. Acute trauma
2. Bronchostenosis with functional distal lung
   A. Tuberculous stricture
   B. Nontuberculous stricture
      1. Traumatic
      2. Inflammatory { Extrabronchial / Endobronchial }
3. Tumors
   A. Bronchogenic cyst
   B. Adenoma
   C. Carcinoma
      1. Hilar with respiratory insufficiency
      2. Tracheal or carinal lesions
      3. Right upper lobe lesions
4. Congenital partial agenesis?

but have no record of, an authenticated instance of a similar situation which could be produced by a nontuberculous, localized, endobronchial infection. The third category, namely that of neoplastic processes, includes several subsidiary divisions. Any type of neoplasm involving the trachea itself allows one but little choice except to utilize some type of graft, or graft plus prosthesis. In fact, it was a tracheal tumor, which had been observed to exist for over two years which incited Daniel's[1] initial interest in this problem. Both primary carcinomas and adenomas of the trachea and bronchial wall have been managed in this manner. We have had opportunity to utilize this approach in the management of a large bronchogenic cyst arising from the posterior tracheal wall. It has also been used in order to resect a more adequate margin of bronchial or tracheal tissue adjacent to neoplasms. Grafts have been used to reconstruct major defects produced by resection of the carina, tracheal wall, and contralateral bronchial wall produced by radical right total pneumonectomy for carcinomas arising in the region of the right upper lobe orifice. Finally, we have found a very definite usefulness of the bronchial graft principle in the management of carcinomas arising in the hilar area of a pulmonary lobe in patients whose respiratory capacity is insufficient to allow any more extensive resectional procedure than lobectomy.

THERAPEUTIC EXPERIENCES

At the present time we have had opportunity to employ grafting procedures in 10 nontuberculous patients. The basic data relative to these 10 patients is briefly summarized in Tables IIA and IIB.

The Use of Grafts in Childhood.—In June, 1951, an 11-year-old boy (W.W.) was referred to us for evaluation of his "bronchial asthma." The history revealed that a metal carpet tack was aspirated at the age of 1 year and was not removed until eighteen months thereafter. For the ensuing eight years he had been noted to have progressive symptoms of recurrent wheezing, mild dyspnea, and recurrent cough. The right lung aerated very poorly upon auscultatory examination. On fluoroscopic and roentgenographic examination (Fig. 1, *A*) a severe obstructive emphysema of the entire right lung, with marked resultant mediastinal shift, was noted. Bronchoscopic

TABLE IIA. SUMMARY OF CLINICAL EXPERIENCES WITH THE USE OF DERMAL GRAFTS IN OPERATIONS NOT INCORPORATING RADICAL RIGHT TOTAL PNEUMONECTOMY

	AGE	DIAGNOSIS	PROCEDURE	RESULTS
L.F.	4	Bronchogenic tracheal cyst	Local tracheal resection and dermal graft	Asymptomatic 16 months
W. W.	11	Benign stricture right main-stem bronchus	Excision and plastic reconstruction with dermal graft	Asymptomatic 32 months
S. H.	52	Adenoma of carina and left main-stem bronchus	Local resection of carina and bronchus with graft	Asymptomatic 7 months
J. C.	48	Extensive right upper lobe carcinoma with severe emphysema	Resection right upper lobe and adjacent tracheal wall with graft. Postoperative radiation therapy	No recurrence 9 months
P. H.	58	Left lower lobe carcinoma with pulmonary fibrosis and emphysema	Resection left lower lobe and medial portion left main-stem bronchus with graft	No recurrence 5 months

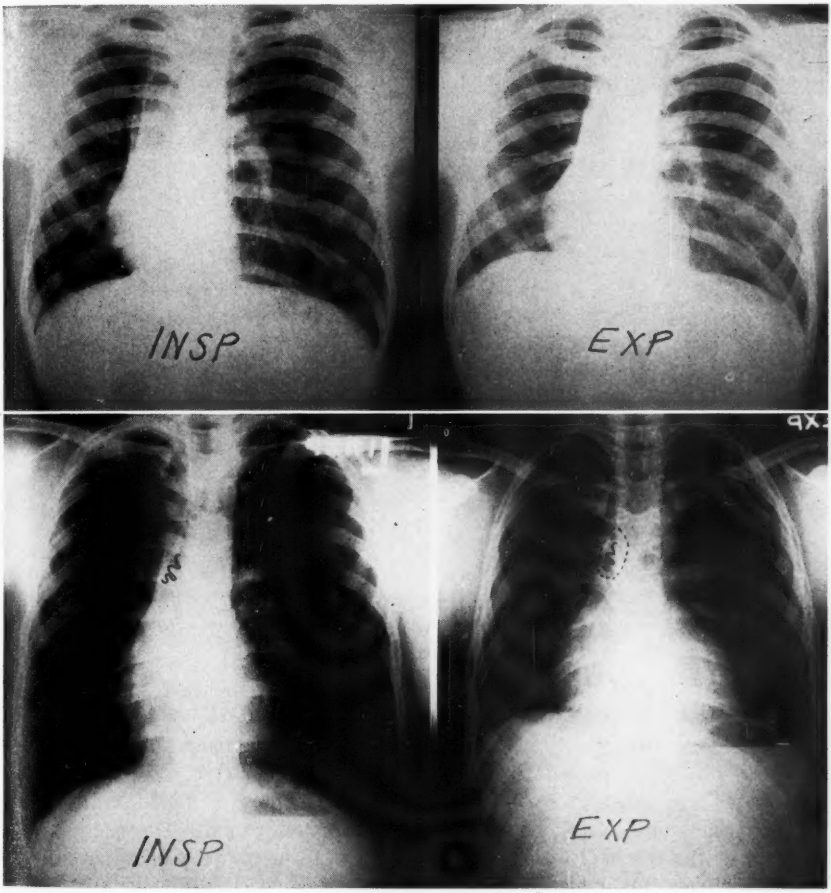
TABLE IIB. SUMMARY OF CLINICAL EXPERIENCES INVOLVING MASSIVE TRACHEOBRONCHIAL DERMAL GRAFTS USED WITH RADICAL TOTAL PNEUMONECTOMY FOR EXTENSIVE RIGHT UPPER LOBE CARCINOMA

	AGE	DIAGNOSIS	PROCEDURE	RESULTS
J. G.	43	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Bronchopleural fistula and empyema-healed: palliative with distal metastasis
T. R.	48	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Bronchopleural fistula, empyema and local recurrence—death 5 months postoperatively
N. W.	49	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Death third postoperative day
G. S.	54	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Delayed empyema and secondary fistula. No recurrence 14 months
G. W.	59	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with glass prosthesis and pleural graft	Local recurrence and distal metastasis. Death 9 months postoperatively

examination revealed a firm, fibrous stricture of the right main-stem bronchus at the level of the orifice of the right upper lobe bronchus. No definite lumen could be visualized. In August, 1951, he was operated upon in the face-down position through a right, high posterolateral incision. Except for some generalized emphysema, the right lung appeared to be healthy. The stricture area was exposed and a classical Gebauer type of operation carried out. The stricture was found to be approximately 6 mm. in length and resembled considerably the type of obstruction seen in coarctation of the aorta. A small graft, approximately 2 by 1 cm. in size, was utilized. Prior to placement of the graft, the fibrotic area of stricture was excised down to healthy cartilage and the cut edges of the bronchial mucosa were approximated by interrupted, inverted, submucosal sutures of No. 0000 arterial silk. Prophylactic tracheotomy was not used, and the postoperative recovery was totally uneventful. The area of bronchial repair has not been visualized by bronchoscopy. The patient has been carefully followed for thirty-two months and subjected to periodic cinefluoroscopic and other examinations. There have been no clinical or radiological evidences to suggest any recurrence of stricture. The right

lung is a little smaller than the left so that slight shift of the heart toward the right on inspiration (Fig. 1, *B*) is still noted, but this continues progressively to decrease. This patient shows no evidences of decrease in respiratory reserve, and his maximum breathing capacity is within normal limits for this age. We feel justified in presuming that the grafted and reconstructed area of the right main-stem bronchus has been able to grow in a satisfactory manner with the rest of the tracheobronchial tree.

A.



B.

Fig. 1.—(W. W.) *A*, Pre-operative inspiration and expiration roentgenograms demonstrating (1) decreased volume on inspiration of right lung and (2) shifting of mediastinum back toward the midline on expiration. Changes due to a severe benign stenosis of the right main-stem bronchus. *B*, Inspiration and expiration roentgenograms thirty-two months after surgery, demonstrating (1) increased volume of right lung on inspiration and (2) essentially normal expiratory function of right lung after surgical correction of bronchial stenosis with a Gebauer type of dermal graft.

In December, 1952, a 4-year-old child (L. F.), was referred to us, again with the impression of "bronchial asthma." This child had had recurrent attacks of pneumonia for a 3½-year period and many episodes of very severe dyspnea. A tendency to very severe dyspnea and wheezing in response to any respiratory tract infection had been noted since birth. The patient presented definite clinical evidence of partial tracheal obstruction. On roentgenological examination (Fig. 2), a large mass could be seen to be present between the trachea and esophagus producing considerable compression and distortion of both organs. On Dec. 15, 1952, with the patient in the lateral position, a high posterolateral incision was made. Preliminary bronchoscopy had not been carried out because of the degree of compression of the trachea known to be present and the fear that any trauma incident to bronchoscopy might produce swelling sufficient to totally occlude the air passages. We

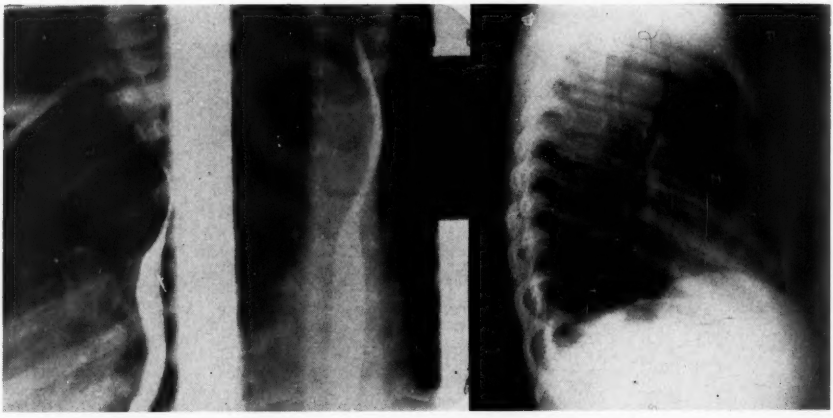


Fig. 2.—(L. F.) Preoperative x-ray studies demonstrating posterior tracheal mass producing major compression and distortion of both esophagus and trachea. Postoperative inspiration and expiration studies sixteen months later showed normal pulmonary function and no evidence of recurrence of the original bronchogenic cyst of the posterior tracheal wall.

had not anticipated the need for resection of the posterior tracheal wall or we would have utilized the face-down position. The technical difficulty of the procedure was greatly increased by the lateral approach. A Portex endotracheal tube was passed beyond the area of tracheal compression before positioning the patient. Exploration revealed a large, firm-walled, multilocular cystic mass, which later proved to be a bronchogenic cyst. The membranous portion of the tracheal wall was so thinned as to be actually transparent and slight tears in this structure rapidly became major-sized defects. The tissue obviously was inadequate and, therefore, a classical Gebauer dermal graft, 2.5 cm. long by 1 cm. wide, was placed. Because of considerable chronic bronchopulmonary suppuration, a prophylactic tracheotomy was carried out at the conclusion of the procedure. Some adherent exudate formed in the region of the graft, requiring bronchoscopy on the second postoperative day.

This procedure was well tolerated. In order to prevent the tracheotomy tube from impinging upon the grafted area, a specially shortened tracheotomy tube was utilized. His postoperative course was otherwise uneventful; the tracheotomy was removed on the fourteenth postoperative day, and he has been totally asymptomatic for the past sixteen months. There has been no recurrence of wheeze or asthmatoid behavior. Repeated roentgenographic examination has shown no evidence of tendency to recurrence of the diffuse obstructive emphysema so prominently noted before operative intervention. The child has grown well, and we presume that the reconstructed area of the trachea has grown commensurately.

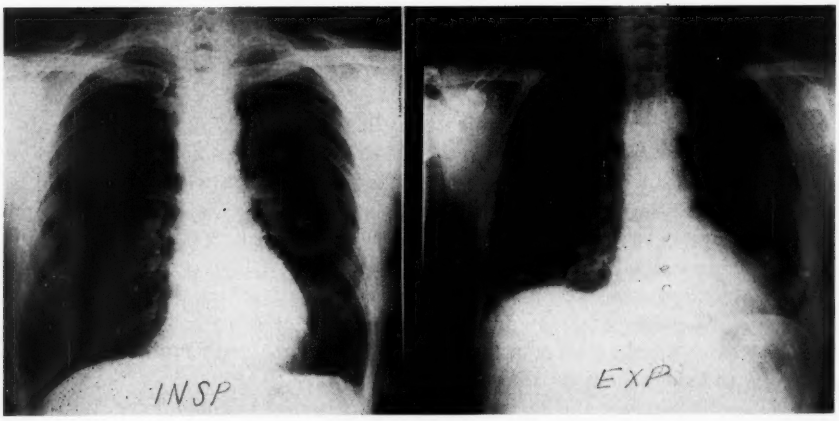
Adenoma of Carina and Left Main-Stem Bronchus.—We have had opportunity to resect adenomas involving the carina on two occasions. The first patient was reported in a previous communication[13] and referred to in the introduction to this publication. The extensive nature of the adenoma throughout the right main-stem bronchus, as well as the carina, required that she be treated by a total pneumonectomy and excision of the carina and adjacent tracheal and left bronchial wall. It was possible to close the defect without graft.

In July, 1953, a 52-year-old man presented himself with a history of excessive smoking for thirty-two years, chronic cough, frequent colds without evident suppuration, and a recent, rather copious hemoptysis. Investigation revealed that he had been conscious of a wheeze over his left chest in association with respiratory tract infections for the past few months. Such a wheeze was demonstrable on auscultation over the left main-stem bronchus. Roentgenograms revealed large, calcified hilar nodes and obstructive emphysema involving the left lung. Laminographic studies (Fig. 3, *A*) revealed a mass in the upper portion of the left main-stem bronchus not completely occluding the orifice. On bronchoscopic examination a typical adenoma was seen occupying the upper medial surface of the left main-stem bronchus and extending to the level of the carina. There was no evidence of suppuration distal to the adenoma. On Sept. 14, 1953, operation was performed with the patient in the face-down position utilizing the high left posterolateral approach. It was necessary to mobilize the aortic arch. The posterior wall of the left main-stem bronchus was incised below the level of the adenoma and the tumor removed with an adequate margin. Adequacy of excision was confirmed by a frozen section study of the cut edges. It was necessary to resect the carina and the defect was reconstructed with a 3.5 by 1.75 cm. dermal graft. Pathologic examination revealed a typical adenoma without evidence of carcinomatous degeneration. No postoperative radiation therapy was utilized. The patient's postoperative course was uncomplicated. He has been totally asymptomatic for the past seven months, and roentgenologic studies reveal the left lung to have a good ventilatory capacity (Fig. 3, *B*).

Hilar Carcinoma With Respiratory Insufficiency.—There have been two instances in which patients presented with resectable tumors which had extended from the point of origin in the hilar area of the involved lobe into the lumen of the main-stem bronchus. In both of these patients a severely de-

pleted respiratory reserve forbade any consideration of total pneumonectomy. It was, therefore, decided that the patients should be treated by lobectomy, radical excision of hilar and mediastinal nodes, and removal of a sufficient margin of the adjacent main-stem bronchus to allow eradication of neoplastic tissue. The resultant defect in the bronchial wall would be repaired by an adequate-sized dermal graft. The first patient (J. C.) was operated upon in July, 1953. He was shown to have a carcinoma of the right upper lobe bronchus which had extended for a short distance into the lumen of the right

A.



B.

Fig. 3.—(S. H.) *A*, Tomographic study demonstrating endobronchial tumor in left main-stem bronchus, near level of carina. Surgical excision required concomitant removal of the carina for adequate margin beyond a carcinoid type of adenoma. *B*, Postoperative inspiration and expiration films seven months following surgical excision of the adenoma and reconstruction of the bronchial and carinal defect with a Gebauer graft.

main-stem bronchus. A roentgenogram (Fig. 4, *A*) revealed that the contralateral, or left upper lobe, was replaced with large emphysematous blebs, and there was some generalized hypertrophic emphysema. His maximum breathing capacity was 49 L. per minute, and it was felt that total right pneumonectomy would produce unjustifiable respiratory limitations. Right upper lobectomy, with resection of the adjacent wall of the trachea and right main-stem bronchus, along with radical gland resection, was performed. The defect was reconstructed with a rather large graft, 5 by 3 cm. over all. Because of the size of the graft, it was desired to have constant access to tracheal secretions in a

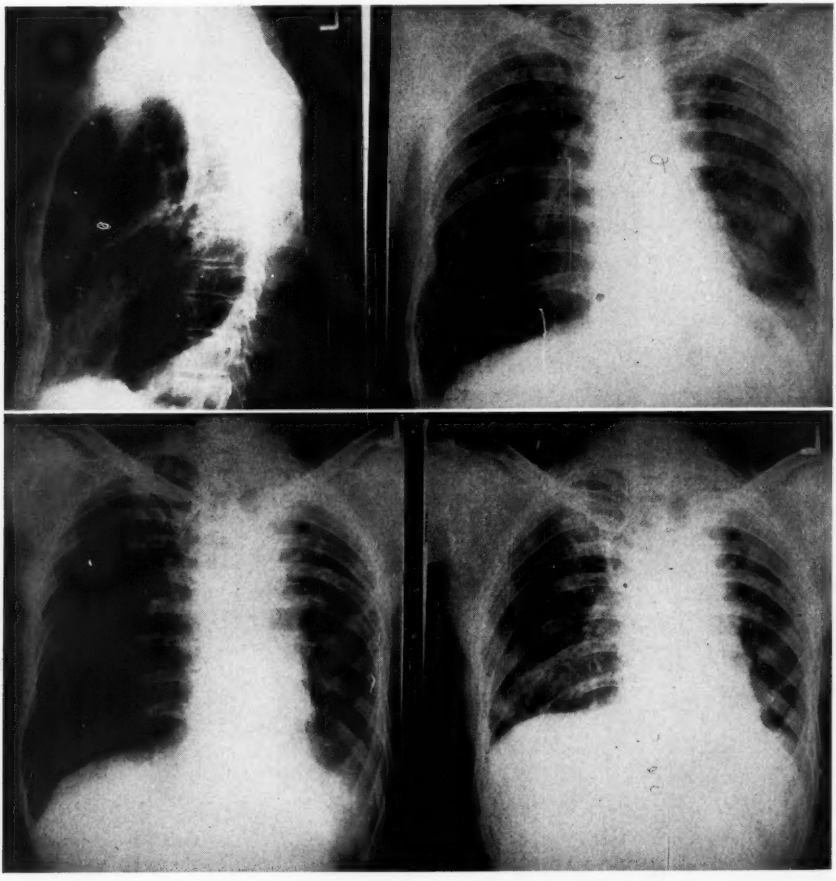
A.

B.

Fig. 4.—(J. C.) *A*, Preoperative chest x-ray demonstrating right upper lobe mass in a patient with bilateral combined bullous and hypertrophic types of pulmonary emphysema. *B*, Postoperative inspiration and expiration roentgenograms following radical-type right upper lobectomy with excision of adequate margin of adjacent wall of trachea and intermediate bronchus. Defect reconstructed with a Gebauer type graft.

patient with respiratory insufficiency, and a prophylactic tracheotomy was carried out. Postoperative course was uneventful, and the tracheotomy was removed on the twelfth postoperative day. A study of the resected lesion revealed the tumor to be quite anaplastic and, therefore, four weeks following operation he was given intensive deep x-ray therapy to the area of the lower trachea, right main-stem bronchus, and mediastinal node region. The patient has been totally asymptomatic for the ensuing nine months. There has been no evidence of recurrence of the disease. Roentgenological examination

A.



B.

Fig. 5.—(P. H.) *A*, Preoperative chest roentgenogram showing extensive bilateral pulmonary fibrosis and complete atelectasis of the left lower lobe. Bronchoscopy revealed an extensive bronchogenic carcinoma of the left lower lobe bronchus. *B*, Postoperative inspiration and expiration roentgenographic studies five months after surgery, demonstrating satisfactory ventilatory function, after plastic reconstruction of left upper lobe and left mainstem bronchus.

(Fig. 4, *B*) would suggest that there has been no progression of the hypertrophic emphysema noted in the right middle and lower lobes prior to operative intervention.

In November, 1953, a 58-year-old man (P. H.) was found to have carcinoma arising in the region of a long-standing area of bronchostenosis in the left lower lobe bronchus. The neoplastic process had extended upward and medially along the bronchial wall to a level parallel to the orifice of the left upper lobe bronchus. Severe associated chronic pulmonary disease, consisting of intensive pulmonary fibrosis and emphysema (Fig. 5, *A*) with a markedly decreased maximum breathing capacity, precluded any consideration of total pneumonectomy. Therefore, on Nov. 16, 1953, utilizing a left lateral approach, the entire left lower lobe, hilar and mediastinal nodes, and an adequate margin of the left main-stem bronchus were resected. A graft 3.5 cm. long and approximately 2 cm. wide was utilized to reconstruct the airway into the left upper lobe bronchus. No major evidence of suppurative tracheobronchial disease was present and, because only a moderately large graft had been placed, prophylactic tracheotomy was not instituted. Because of the severe degree of pulmonary fibrosis, postoperative x-ray therapy was not given. The patient has been active, without evidence of recurrence of disease, for five months. Periodic fluoroscopic and physical examinations have indicated satisfactory ventilatory capacity of the remaining lung tissue on the left. (Fig. 5, *B*).

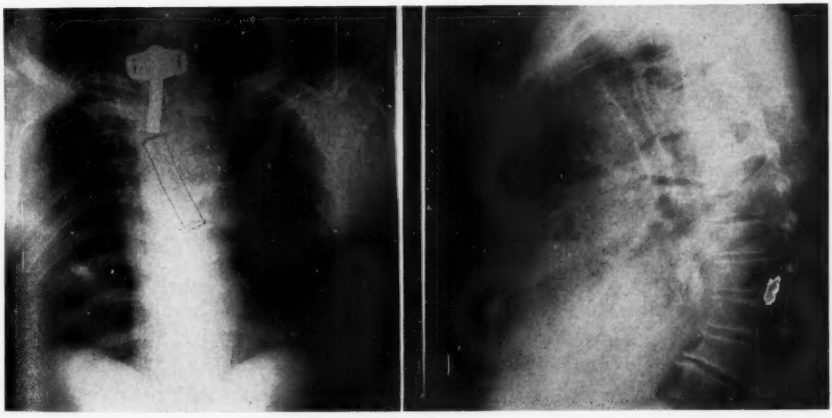


Fig. 6.—Roentgenograms showing glass prosthesis supporting an extensive pleural graft used to reconstruct a surgically produced massive tracheobronchial defect produced during radical right total pneumonectomy for right upper lobe bronchogenic carcinoma.

Extensive Bronchogenic Carcinoma Arising in the Region of the Right Upper Lobe Orifice.—We have had opportunity to carry out total right pneumonectomy with associated radical mediastinal gland excision and concomitant extensive resection of the adjacent tracheal wall, carina, and medial wall of the left main-stem bronchus in ten patients. The resultant defect was replaced by some type of graft in five instances. In one patient, in whom the excisional

defect measured approximately 10 cm. in length, a flanged Pyrex glass tube (Fig. 6) was used for reconstruction. Absorbable sutures were placed about the flanged area and brought out through the tracheobronchial wall at three points, both above and below the defect, to keep the prosthesis in place. A double thickness pedicle pleural graft was then sutured over the defect so as to make it airtight. Prophylactic tracheotomy was not performed. This operation was carried out in May, 1948, and the hazards of removal of such a prosthesis had not been described. Two months following surgery the patient was readmitted to the hospital and, through a cervical tracheostomy, the prosthesis was removed. Stenosis of the reconstructed area slowly but progressively ensued. The patient succumbed nine months after initial operative procedure due to local and generalized recurrence of carcinoma. During the initial two months following surgery, when the prosthesis was still in place, the patient had been distinctly palliated. Following removal of the prosthesis and progressive bronchial stenosis the patient was essentially totally debilitated. It is now felt that the tube could safely have been left in place, and the palliation and temporary rehabilitation would have fully justified the operative procedure. Following our experiences with this patient, we awaited the advent of the Gebauer dermal graft before subjecting further similar patients to this type of operative procedure. During the past fourteen months, we have had opportunity to operate upon four additional patients with extensive carcinoma of this type. The first such patient (G. S.) was operated upon and the defect reconstructed with a graft 6 cm. in length and 3 cm. in width. Prophylactic tracheotomy was not instituted. The patient's postoperative course was uneventful. Constant major attention was paid to the positioning of the mediastinum and to the pressures presenting in the right pleural space. An attempt was made to try to keep the mediastinum slightly to the left of the midline. Not long after his discharge from the hospital the patient began to run a low-grade fever. He lived at a considerable distance and did not return to us for further evaluation until three months after the initial operative procedure. By this time an extensive empyema of the right pleural space had been produced and could not be controlled by aspiration measures. This intrapleural infection produced a progressive and essentially complete necrosis of the grafted area, with resultant bronchopleural fistula. External drainage was necessary, at which time a foreign body was discovered in the pleural space and removed. The patient has been maintained on adequate drainage, and, despite the metastatic involvement of deep mediastinal nodes removed at the time of surgery, he now presents no evidence of recurrence of disease fourteen months after the initial procedure. It is our plan to carry out a modified Roberts[17] type thoracoplasty in the near future. A tantalum mesh sheet will be incorporated in the area of persistent fistula, over which will be placed the overlying intercostal muscle bundles. We feel confident that this patient would have had major rehabilitation along with possible eradication of his disease had he not been subjected to a delayed and unnecessary empyema.

Twelve months ago, a 43-year-old male veteran (J. G.) with a similar right upper lobe carcinoma was operated upon, and a graft approximately 5

by 3 cm. was utilized. He developed a small bronchopleural fistula six days following operation and was treated by thoracotomy drainage and tracheotomy. Two months later the empyema space and fistula were eradicated by means of a Roberts type of thoracoplasty, plus tantalum mesh coverage of the fistulous area. The patient responded well to this operative procedure and, for five months, appeared to have major benefit. He then presented with a large abdominal mass, representing a massive metastatic involvement of one kidney. He was given intensive radiation and is still surviving, with considerable persistent residual tumor.

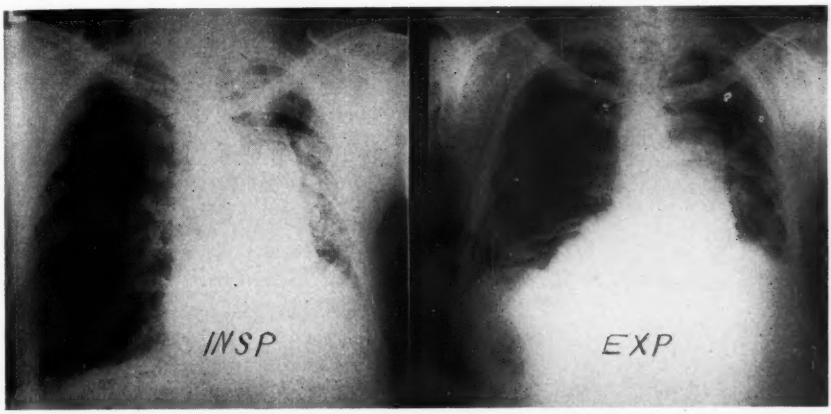


Fig. 7.—(N. T. W.) Inspiration and expiration roentgenographic study to demonstrate almost complete atelectasis of right lung, marked shift of mediastinum and heart to the right, and associated obstructive emphysema of the left lung. (Note: Films printed in reverse.)

Another of these extensive right upper lobe carcinoma patients deserves brief mention. The patient (N. T. W.), a 49-year-old man, presented himself in March, 1954, with a history of cough, hemoptysis, weight loss, and dyspnea. Radiological examination (Fig. 7) showed a very severe degree of obstructive emphysema bilaterally with almost no air exchange in the right lung. Bronchoscopy revealed the right main-stem bronchus to be totally occluded with a neoplastic process spreading across the carina on to the medial wall of the left main-stem bronchus (Fig. 8). The functional opening into the left main-stem bronchus would have admitted approximately two paper matchheads. Operation was performed, at which time it was not possible to get the long tracheotomy tube into the left main-stem bronchus prior to positioning the patient, but an adequate air exchange was maintained with high positive pressure. On opening the chest and slight manipulation of the lung, immediate severe restriction of ventilation in the left lung occurred. A good airway could be maintained while the thoracic trachea could be exposed, tracheostomy performed, and a Portex intratracheal tube (Fig. 9) inserted directly past the occluding neoplastic tissue on the left. A radical excisional procedure, including total pneumonectomy, radical mediastinal gland resection, and extensive excision of tracheal wall carina and medial wall

Fig. 8.

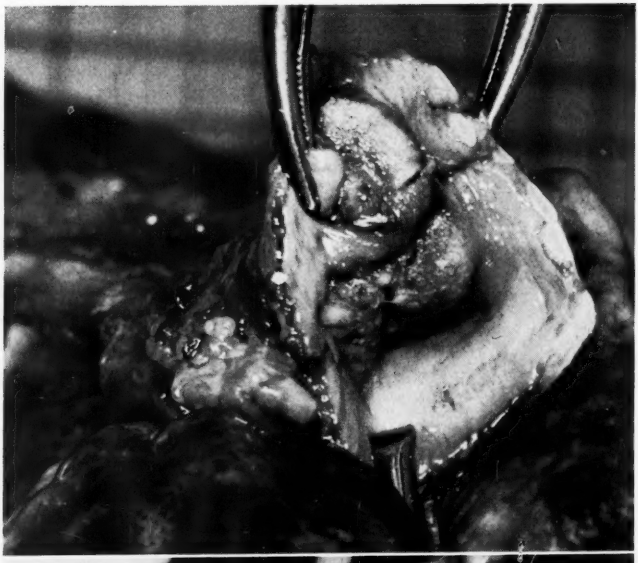


Fig. 9.

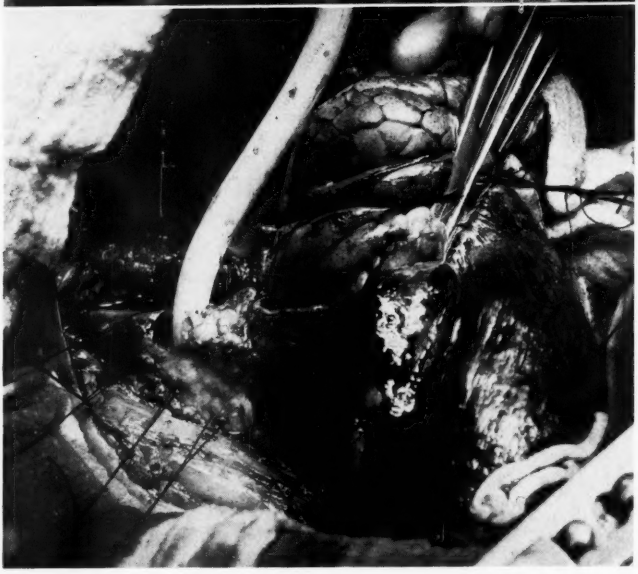


Fig. 8.—(N. T. W.) Resected carina and right lung showing gross extension of tumor into left main bronchus and obstruction of right main bronchus by an extensive bronchogenic carcinoma arising from the orifice of the right upper lobe. The two upper clamps grasp the edges of the left main-stem bronchus.

Fig. 9.—(N. T. W.) Photograph taken during surgery to demonstrate that the "operating team's" Portex tube has been inserted through lateral wall of trachea directly into the left main bronchus to maintain airway while right lung and carina are removed.

left main-stem bronchus was performed. A large graft (Fig. 10) was utilized to reconstruct the defect, utilizing No. 26 silver wire which provided much more adequate support. A very small strand of lower trachea and left main-stem bronchial tissue persisted so that approximately 75 per cent of the airway had to be reconstructed. A Portex tube was used for the prosthesis and sewn in place. This type of prosthesis proved to be deficient in two main categories: (1) the thickness of its wall too greatly decreases the functioning lumen; (2) the rigidity of the wall is such as to produce a major tendency for

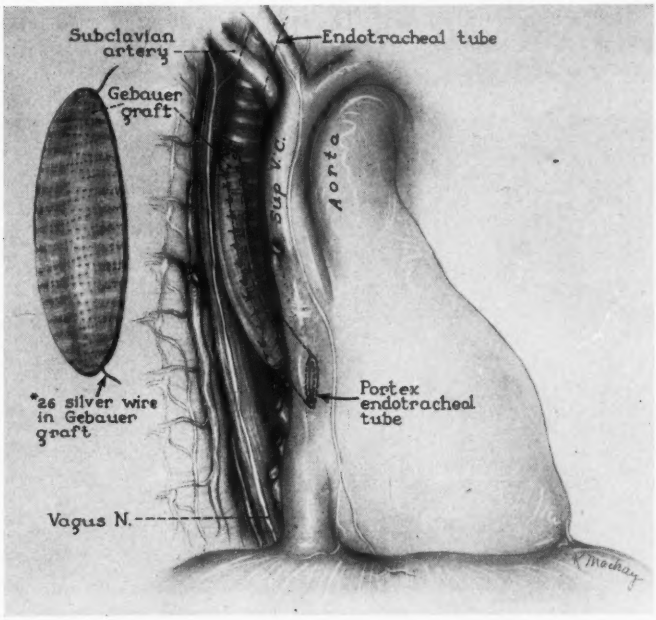


Fig. 10.—Artist's drawing which demonstrates the size of the area of the trachea, carina, and medial wall of the left main-stem bronchus excised with radical right total pneumonectomy. Inset on the left designates the size and shape of the Gebauer type dermal graft utilized. Note that No. 24 or No. 26 silver wire is used in graft replacing areas previously supported by cartilaginous rings.

the upper portion of the tube to lie against the right lateral wall of the trachea above the defect and thus produce obstruction. This patient had considerable repetitive obstructive phenomena during the first few hours following surgery, and this was overcome by placing a No. 4 Portex endotracheal tube into the lumen of the upper stump of the prosthesis through the tracheotomy opening which had been made immediately following the operative procedure. The patient showed gradual improvement, but died suddenly on the third postoperative day. Postmortem examination could not be obtained. Death was probably due to accumulated secretions with obstruction. At the time of surgery considerable measurements were made and consideration was given to the possibility of bringing the stump of the left main-stem bronchus upward and implanting it upon the trachea. This would have involved an upward dis-

placement of over 10 cm. and appeared to be totally nonfeasible. We feel that this patient was very instructive. He did show a reasonable method of management of the airway during operation. He further showed some of the dangers of an inadequate prosthesis. A curved polyethylene tube with thin wall would have been preferable. If a Portex tube had been utilized, he should have kept the long tube which is used for initial reconstructive procedures and, at the end of the operation, had it brought out through a tracheotomy opening so that it simulated a long plastic tracheotomy tube capable of spanning the defect. This method was used on one other patient and appeared to show some definite usefulness. The type of apparatus used on this final patient (T. R.), a 48-year-old man, is illustrated in Fig. 11. This patient had a long graft, 7.5 cm. in length, but only 2.5 cm. in width.

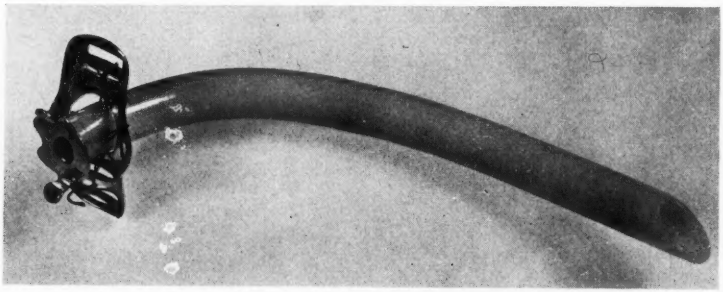


Fig. 11.—(T. R.) Portex tube with tracheotomy tube guard and inner tube used through tracheostomy to provide temporary support for graft and to maintain an adequate airway capable of removal, cleansing, and reinsertion.

This patient developed a small bronchopleural fistula requiring external drainage, with some loss of the graft substance. He had progressive difficulty with narrowing of the main-stem bronchus beginning approximately three months after surgery, and he succumbed five months after surgery because of major local and general recurrence of the disease.

ANESTHETIC AND TECHNICAL CONSIDERATIONS

The major considerations in the line of anesthesia, of course, consist of adequate oxygenation and ventilation. For this purpose the patient should be initially intubated with the extra long intratracheal tube. A Portex tube without cuff is adequate if it is of sufficient size to fit snugly into the left main-stem bronchus. On some occasions we have utilized a long McGill catheter with a balloon. One must always have an extra long intratracheal tube at hand which has been sterilized and can be placed through a transpleural tracheostomy at any time indicated. It is mandatory that such an intratracheal tube be available at all times. This tube may be used as a temporary measure and connected directly to the anesthetic machine. While this transtracheostomy tube is being utilized, the original catheter may be inserted by the anesthetist and then passed across the defect under direct vision

by the operator. This catheter may be left in place after complete repair of the defect and closure of the chest. A tracheotomy is then performed. The Portex tube is transected at a height sufficient to allow it to be brought out through the tracheotomy wound, which allows us to have produced a long, removable tracheotomy tube made of pliable material, which passes beyond the area of defect. It is not thought that this type of prosthesis is necessary except in major-sized grafts. It is our impression that grafts 5 cm. in length or shorter, with adequate intrinsic support, do not necessarily need prosthetic supports. We would also like to emphasize the importance of the supporting sutures about the trachea and bronchus which were emphasized by us in an earlier communication, and which are again illustrated in Fig. 12. This maintains a good support of the mediastinum and helps to hold open the airway,

Fig. 12.—Photograph taken during operation which demonstrates (1) the supporting sutures in the trachea and bronchus used to stabilize the mediastinum, and (2) the Portex tube in place maintaining the airway during grafting procedure.

particularly while manipulations such as changing or moving the long intratracheal tubes are being carried out. We have found a very marked advantage in utilizing the Rand-Wolfe Respirator, since if high pressures are instituted with this machine, for example, 20 to 25 cm. of water, adequate aeration of the contralateral lung can be carried out even in the face of definite large bronchial leaks. The delivered pressure to the lung parenchyma is much below the recorded pressure on the machine due to these leaks. The pressure is graduated according to the position of the mediastinum and the patient's needs. In regard to tracheotomy as a postoperative procedure, this is decided fundamentally by the amount of suppuration present in the individual patient and by the size and character of the graft utilized.

In regard to technical operative considerations, we certainly concur very fully with Gebauer[17, 18] in the benefits of the face-down position in the man-

agement of lesions of the posterior tracheal wall and of the left main-stem bronchus. The lateral position is quite satisfactory for the radical total pneumonectomy and lobectomy procedures. The preparation of the graft must assiduously follow the detail outlined by Gebauer. We have found it very useful to have a moderate-sized block of balsa wood, which facilitates the anchoring of the graft with the straight pins. We would like to emphasize strongly the need for a paper pattern to be sterile and available. This is placed about the endotracheal tube or prosthesis so that adequate graft to cover the curved contour will be produced. It is very important that the graft be cut distinctly larger than the paper pattern. The paper pattern should be kept in the patient's chart to aid in further investigative study. It is important to stress, in the radical pneumonectomy group where approximately three-fourths of the circumference of the trachea and bronchus is being removed, that very adequate support of the wall of the graft is mandatory. Our experience has shown us that, while we agree with Gebauer in the desirability for relatively fine silver wire supporting sutures in the graft when repairing stenotic lesions and just replacing the soft membranous portion of the trachea or bronchus, a considerably different situation is presented when we are replacing major-sized areas of trachea or bronchus previously supported by a cartilaginous framework. For this latter purpose, we prefer to use a No. 26 or even a No. 24 silver wire suture for the internal support of a considerably thick-walled graft. In the repair of stenotic lesions and replacement of noncartilaginous tracheal or bronchial tissue, the No. 28 or No. 30 silver wire is fully adequate. In the repair of stenotic lesions it is necessary to utilize molds to curve the graft properly. The replacement of major portions of the cartilaginous tracheal wall utilized the indwelling tracheal tube for a prosthesis in place of the mold to aid in the proper curvature of the replacement tissue. Probably the most common error which we have incurred is to obtain grafts of inadequate width. It is important to plan a graft which, if it overlies a tube or prosthesis, will have some definite redundancy. Compression upon the graft by a prosthesis, permanent or temporary, produces compression phenomena unfavorable to the circulation. Furthermore, a graft which is too snug has a marked tendency to flatten and produce an inadequate tracheobronchial lumen. It is obvious from the preceding discussion that we do not feel that a completely adequate prosthetic substance is at hand and further measures must be carried out to study this problem. Our present preference is for a thin, curved polyethylene tube. It is necessary for such a tube to have a distinct difference in the diameters of its upper and lower orifices. We have, on all occasions, utilized a large pedicle pleural graft which is sewn over the dermal graft or over an exposed prosthesis in one instance. This is necessary in order to seal the airway. We initially used to place Gelfoam between the pleural and dermal graft, but now feel that this decreases potential blood supply for larger grafts. Grafts, 3 cm. in length or smaller, are carried out in the classical Gebauer manner with the interspersed Gelfoam. Grafts of greater length, and particularly grafts of greater width, are felt to require a source of external blood supply, and we prefer to employ a pedicle graft, utilizing the pleura and fat from the pericardial region because of its

vascularity. Following completion of the graft, the pleural cavity is filled with saline to test the pleura and ensure the presence of a completely airtight reconstruction. In cases of total pneumonectomy a thoracotomy tube is placed through the third anterior interspace. This tube is not utilized for drainage, but is clamped an adequate distance from the chest and the clamp handles seal-closed with adequate adhesive strapping. This tube is utilized for frequent measurements of the intrapleural pressures and, if necessary, to serve as a source for injection of either air or plasma to help splint the mediastinum in the proper position.

We do not feel that there need be any major fear of bronchoscopic maneuvers following operative procedures. They have been done frequently with our grafts and have been reported by others. They are useful for the removal of exudate, debris, and granulation tissue should it occur. Certainly bronchoscopy is not justified in these procedures except upon therapeutic indications. If a prophylactic tracheotomy is in place, this opening is utilized for the bronchoscopic approach. If bronchoscopy is required following surgery and the patient does not have prophylactic tracheotomy, it is usually felt advisable to institute the tracheotomy at that time.

FUNDAMENTAL PROBLEMS

There are certain fundamental problems which have been brought forward by experiences in this field. The first is whether or not the procedure is justified. Our experience indicates that it is fully justified in all the circumstances initially set down, with the reservation that there is some doubt relative to its justification in extensive right upper lobe carcinomas which have invaded beyond and across the carina. It is our feeling that, despite our present major complication and minimal palliation rate outlined in our above experiences with these lesions, an open mind should be kept. Further investigative experiences of laboratory and clinical types should be studied. It is our impression that, if we will insist upon a more adequate width of graft and the firmer support presented by No. 24 silver wire, some of the complications inherent in the placement of massive grafts in the reconstruction of cartilaginous areas of the tracheobronchial wall will be eradicated. There remains a distinct problem in regard to how large a graft one can place before too high an incidence of partial necrosis and fistula is produced. It is our present feeling that grafts 5 cm. in length and 2.5 cm. in width have been proved to survive, while those over 5 cm. in length, with a width of 3 cm. or greater, stand a high chance of local necrosis. Therefore, in the larger grafts an underlying prosthesis should be utilized. The exact type of prosthesis is not as yet defined, but our present trend is toward a specially constructed and perforated polyethylene tube.

The problem of the need of postoperative tracheotomy is also an important question. Certainly there is some benefit in decreasing the pressure produced upon the suture line by cough with the closed glottis and intact cervical tracheal wall. The tracheotomy does distinctly introduce a certain hazard of potential infection of the graft and restricts the normal humidification of the tracheobronchial airway. Our feeling is that the need for tracheotomy is

an individual factor to be determined by graft size, the presence or absence of a prosthesis, and the suppurative aspect of the patient's tracheobronchial tree.

Initial experiences with employment of grafts in children suggest strongly that small or moderate-sized grafts will grow. One would fear the use of grafts larger than 3 by 1.5 cm. in the younger age group. Further observation and study, particularly with experimental investigation, are needed to define the fate of grafts placed in infancy or childhood.

A further important problem is the consideration of bringing an adequate blood supply to the base of large grafts. Graft survival requirements imply restrictions produced by dimensions and by the blood supply of the remaining strand of tracheobronchial tissue to which it is attached. It would seem reasonable to hope that a fairly vascular, pedicle type, pleural-fat graft, placed directly upon the base of the transplanted dermis, might decrease tendencies to local necrosis of the graft substance.

SUMMARY AND CONCLUSIONS

The background investigations and clinical experiences relative to tracheobronchial reconstruction have been outlined. The value of the contributions of Gebauer, relative to reconstruction of the tracheobronchial tree by grafting and other methods, is emphasized. The indications for tracheal or bronchial reconstruction by grafting procedures have been detailed and fall into three major categories. These consist of the management of acute trauma, of various types of bronchial stenosis, and of neoplastic lesions. Congenital defects may constitute a fourth indication. Experiences with the application of various types of grafting procedures in ten nontuberculous lesions are outlined. Limited experience suggests that grafts are well tolerated in childhood and appear to grow in a satisfactory manner with the child. The use of grafts in the management of bronchogenic cyst of the tracheal wall and of a stricture secondary to prolonged endobronchial trauma by a foreign body have each been carried out successfully. Experiences in two instances show that a more adequate operation for carcinoma arising in the hilar area of a lobe in patients with a severe associated restricted respiratory capacity can be performed. In one instance the graft has successfully tolerated intensive postoperative deep x-ray therapy. An additional experience of management of an adenoma of the left main-stem bronchus and carina, with correction by local resection and grafting, has been presented. It is hoped that the use of bronchial grafts may extend the limits of operability of carcinoma arising in the right upper lobe area so a more adequate excision of tracheal, carinal, and contralateral bronchial tissue may be performed. The limitations and problems inherent in the procedure have been presented.

REFERENCES

1. Daniel, R. A.: The Regeneration of Defects of the Trachea and Bronchi, An Experimental Study, J. THORACIC SURG. **17**: 335, 1948.
2. Taffel, M.: The Repair of Tracheal and Bronchial Defects With Free Fascia Grafts, Surgery **8**: 56, 1940.
3. Longmire, W. P., Jr.: The Repair of Large Defects of the Trachea, Ann. Otol. Rhin. & Laryng. **57**: 875, 1948.

3a. Clagett, O. T., Grindlay, J. H., and Moersch, H. J.: Resection of the Trachea, an Experimental Study and Report of a Case, Arch. Surg. **57**: 253, 1948.
3b. Jarvis, F. J.: Discussion of Gebauer's Article, J. THORACIC SURG. **20**: 649, 1950.
3c. Carter, Max, G., and Strieder, J. W.: Resection of Trachea and Bronchi, An Experimental Study, J. THORACIC SURG. **20**: 613, 1950.
4. Ehrlich, R. W., Meyer, R. P., Taylor, C. B., Hass, G. M., and Miller, E. M.: Reconstruction of the Tracheobronchial Tree With Resection of the Lung, Carina and Lower Trachea, Surg., Gynec. & Obst. **94**: 570, 1952.
5. Graig, R. L., Holmes, G. W., and Shabart, E. J.: Tracheal Resection With Replacement With a Prosthesis, J. THORACIC SURG. **25**: 384, 1953.
6. Gebauer, P. W.: Plastic Reconstruction of Tuberculous Stenosis With Dermal Grafts, J. THORACIC SURG. **19**: 604, 1950.
7. Robb, C. G., and Bateman, G. H.: Reconstruction of the Trachea and Cervical Esophagus, Brit. J. Surg. **37**: 202, 1949.
8. Griffith, J. L.: Fracture of the Bronchus, Thorax **4**: 105-109, 1949.
9. Maisel, B., and Dingwall, J. A.: Primary Suture of the Divided Cervical Trachea, Surgery **27**: 726, 1950.
10. Ferguson, D. J., and Wild, J. J.: Experimental Resection of the Trachea, Surgery **28**: 597, 1950.
11. Mathey, J., and Oustrieres, G.: End-to-end Bronchial Anastomosis After Accidental Division of the Stem Bronchus, Thorax **6**: 71, 1951.
12. Paulson, D. L.: Traumatic Bronchial Rupture With Plastic Repair, J. THORACIC SURG. **22**: 636, 1951.
13. Abbott, O. A.: Experiences With the Surgical Resection of the Human Carina, Tracheal Wall and Contralateral Wall in Cases of Right Total Pneumonectomy, J. THORACIC SURG. **19**: 906, 1950.
14. Gebauer, P. W.: Further Experiences With Dermal Grafts for Healed Tuberculous Stenosis of the Bronchi and Trachea, J. THORACIC SURG. **20**: 628, 1950.
15. Goldman, A.: The Surgical Treatment of Bronchial Adenoma, Dis. Chest **13**: 321, 1947.
16. Langston, H. T., and Fox, R. T.: Indication for Posterior Transpleural Bronchotomy in the Management of Intrabronchial Tumors, Surg., Gynec. & Obst. **86**: 192, 1948.
17. Gebauer, P. W.: Reconstructive Surgery of the Trachea and Bronchi: Late Results With Dermal Grafts, J. THORACIC SURG. **22**: 568, 1951.
18. Gebauer, P. W.: Pulmonary Surgical Salvage by Bronchial Resection, Surg., Gynec. & Obst. **94**: 347, 1952.

(For Discussion, see page 252.)

BRONCHIAL ANASTOMOSIS AND BRONCHOPLASTIC PROCEDURES IN THE INTEREST OF PRESERVATION OF LUNG TISSUE

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DALLAS, TEX.

BRONCHIAL anastomosis and bronchoplastic procedures in the interest of preservation of lung tissue are relatively new developments in the field of thoracic surgery. A traumatic, inflammatory, or neoplastic lesion of the bronchus does not always require resection of all of the lung tissue supplied by the involved bronchus. By means of various types of repair, it is possible to excise a portion of bronchus and to restore continuity, thus salvaging a portion or all of the lung tissue distal to the point of excision.

The reports of Gebauer[1-3] made before this association have demonstrated very well the merit of the concept of pulmonary salvage through bronchial reparative procedures, particularly with reference to tuberculous bronchostenosis but including bronchial tumors. Many others through individual published reports and discussion of Dr. Gebauer's papers have recorded the successful application of various reconstructive procedures of the bronchus following excision of tumors, traumatic strictures, and accidental bronchial division.

The authors have used a variety of procedures to restore bronchial continuity and preserve good lung tissue in sixteen patients. The bronchial lesions so treated include traumatic occlusion (2), acute traumatic rupture (2), tuberculous stenosis (3), adenoma (2), and carcinoma (7). The individual cases are summarized in Tables, I, II, III, and IV with appropriate illustrations.

TABLE I. TRAUMATIC BRONCHIAL RUPTURE

CASE	SEX AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
1. L. M.	M 22	Traumatic bronchial occlusion, right main bronchus, 7 weeks following injury	Plastic reconstruction of right main bronchus by means of dermal graft (Fig. 1)	July 2, 1949	Bronchus patent. Lung fully expanded
2. B. S.	M 21	Traumatic bronchial rupture, left main bronchus, 6 weeks following injury	End-to-end anastomosis of left main bronchus (Fig. 2)	Aug. 8, 1951	Bronchus healed. Lung fully expanded
3. J. C.	M 19	Traumatic bronchial rupture, 6 hours following injury	End-to-end anastomosis, left lower lobe bronchus to left main bronchus (Fig. 3)	April 19, 1951	Bronchus healed. Lung fully expanded
4. B. H.	M 23	Traumatic bronchial rupture, 2 hours following injury	Tracheotomy. Suture of tear involving right main, intermediary, and superior segmental bronchi (Fig. 4)	Jan. 20, 1954	Bronchus healed. Lung fully expanded

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Read at the Thirty-fourth Annual Meeting of The American Association for Thoracic Surgery at Montreal, Quebec, May 3-5, 1954.

TRAUMA

Rupture of the bronchus may result from nonpenetrating trauma of the chest. The diagnosis during the period immediately following the injury can be readily made if one keeps in mind the possibility of a tear of the bronchus in a patient who has sustained nonpenetrating trauma of the chest and in whom a tension pneumothorax and/or mediastinal emphysema rapidly develops. A bronchial tear should be suspected if, following decompression of the tension pneumothorax, air continues to bubble vigorously through the water seal system and if the lung fails to expand. Bronchoscopy done immediately following the injury will reveal in most cases the site of the bronchial tear, as usually it occurs in either main bronchus at or within a few centimeters of the carina. However, secondary bronchi may be torn. There may be either a partial tear or complete transection of the bronchus. A great many of these injuries have ended fatally within a short time following the accident because of failure to make the diagnosis until autopsy revealed the true condition.

Prompt recognition of a traumatic bronchial rupture must be followed immediately by thoracotomy and repair of the torn bronchus, as illustrated in Cases 3 and 4. By means of an end-to-end anastomosis, the left lower lobe was preserved in Case 3. The suture of a large tear involving the right main, intermediary, and superior segmental bronchi resulted in the salvage of an entire lung in Case 4.

If the patient survives the early posttraumatic period, the unrepaired ruptured bronchus heals with either complete occlusion or stricture of the bronchus. Bronchial anastomosis is indicated for complete traumatic bronchial occlusion should such a patient be seen months or even years after the initial injury. Pneumonectomy is not justified unless pulmonary suppuration is present, and this cannot occur in the case of complete occlusion.[4, 5] The collection of thick viscid mucus within the bronchi of the lung blocked by complete bronchial occlusion must not be mistaken for suppuration. The lung itself, although atelectatic, undergoes no pathologic change. A bronchoplastic procedure as carried out in Case 1 or an end-to-end anastomosis as made in Case 2 should be done to relieve the bronchial occlusion and restore bronchial continuity. The atelectatic lung will re-expand and resume function if bronchial continuity is restored, even years after the initial injury. Samson and Evans[6] have recently successfully re-expanded a lung by means of a bronchial anastomosis in a patient fifteen years after a traumatic bronchial occlusion was sustained.

The pulmonary suppuration which will develop if the bronchial tear heals by stricture formation, without complete occlusion, makes imperative the early diagnosis and resection of the traumatic bronchial stricture and restoration of bronchial continuity by end-to-end anastomosis.

By early diagnosis and bronchial reconstructive procedures, the mortality of bronchial rupture can be markedly reduced and lung function preserved.

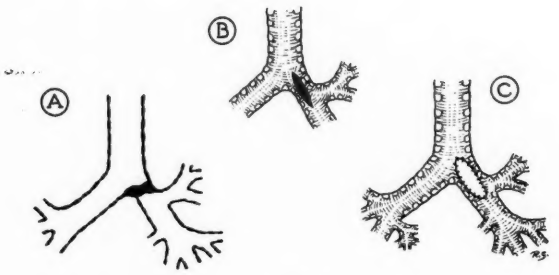


Fig. 1.

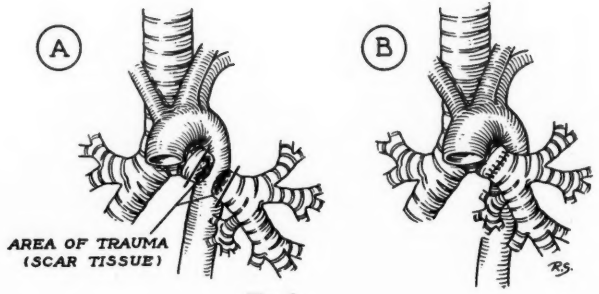
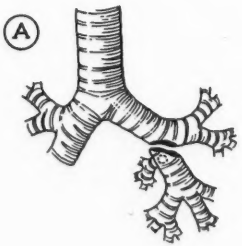


Fig. 2.

Figs. 1-4.—Traumatic bronchial rupture.

Fig. 1.—Bronchial occlusion, right main bronchus. Incision and dermal graft seven weeks after injury. (Case 1.)

Fig. 2.—Complete separation of left main bronchus. Excision of scar and end-to-end anastomosis six weeks after injury. (Case 2.)



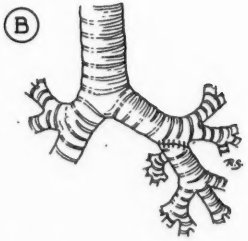
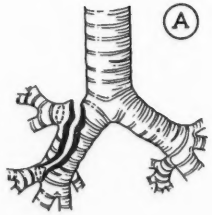


Fig. 3.



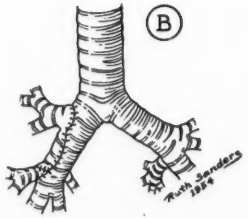


Fig. 4.

Fig. 3.—Complete separation of left lower lobe bronchus. End-to-end anastomosis six hours after injury. (Case 3.)
Fig. 4.—Tear of right main, intermediary, and superior segmental bronchi. Suture two hours following injury.

Fig. 5.

Fig. 6.

Figs. 5 and 6.—Tuberculous bronchostenosis.

Fig. 5.—Healed stenosis, right main bronchus. Right upper and middle lobectomies, incision of stenotic area and reconstruction of dermal graft reinforced by steel wire mesh. (Case 5.)

Fig. 6.—Healed stenosis, right main and right upper lobe bronchi. Resection of right main bronchus and right upper lobe; end-to-end anastomosis. (Cases 6 and 7.)

TUBERCULOUS BRONCHOSTENOSIS

Healed tuberculous bronchostenosis, usually involving the primary and secondary bronchi, may be resected and bronchial anastomoses done. If the stenosis involves the main bronchus and the lower trachea, as in Case 5, a plastic reconstruction by means of a dermal graft reinforced by steel wire mesh may be utilized after incising the stenotic area. A lobectomy or segmental resection may be done at the same time as the bronchial anastomosis or plastic reconstruction. By re-establishing bronchial continuity, the remaining healthy lobes of lung are preserved, as illustrated in Cases 5, 6, and 7. By means of these procedures a total of 5 lobes of lung were preserved in these 3 cases. There were no complications and no mortalities.

TABLE II. TUBERCULOUS BRONCHOSTENOSIS

CASE	SEX AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
5. A. T.	F 20	Pulmonary tuberculosis with 2 cavities persisting following thoracoplasty in 1950. Healed stenosis, right main bronchus	Right upper and right middle lobectomies. Plastic reconstruction of right main bronchus and lower trachea by means of dermal graft reinforced by steel wire mesh (Fig. 5)	Dec. 15, 1951	Bronchus healed. Good expansion of lower lobe. Sputum negative
6. D. C.	F 34	Pulmonary tuberculosis, right upper lobe. Healed stenosis, right main and right upper lobe bronchi	Resection of right upper lobe and right main bronchus. End-to-end anastomosis of intermediary bronchus to trachea (Fig. 6)	July 30, 1953	Bronchus healed. Right middle and lower lobes fully expanded. Sputum negative
7. R. B.	F 31	Pulmonary tuberculosis with destruction of right upper lobe	Right upper lobectomy which compromised the lumen of the right main bronchus. Transverse division of bronchus and end-to-end anastomosis	Nov. 7, 1953	Bronchus healed. Right middle and lower lobes fully expanded. Sputum negative

TABLE III. BRONCHIAL ADENOMA

CASE	SEX AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
8. J. S.	F 24	Bronchial adenoma medial wall of right main bronchus	Wedge excision and transverse suture (Fig. 7)	June 12, 1951	Bronchus healed. Lung fully expanded. Well to date
9. L. H.	M 43	Bronchial adenoma anterolateral wall, left lower lobe bronchus	Left lower lobectomy and excision of adenoma, preserving a flap of bronchial wall used for reconstruction (Fig. 8)	June 2, 1952	Bronchus healed. Left upper lobe fully expanded. Well to date

BRONCHIAL ADENOMA

Because bronchial adenoma is a tumor of low-grade malignancy, it lends itself well to localized resection and bronchial reconstructive procedures in the interest of preservation of healthy lung tissue. Only in the presence of distal pulmonary suppuration is the resection of lung tissue justified. Case 8

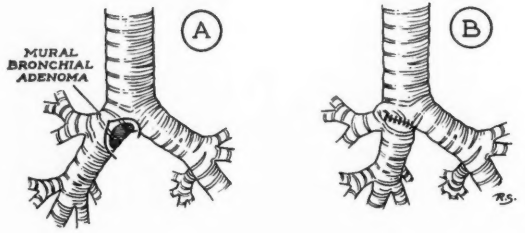


Fig. 7.

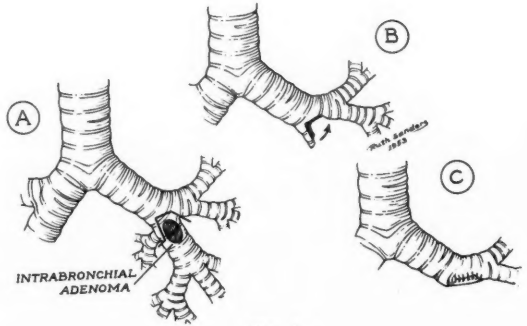


Fig. 8.

Figs. 7 and 8.—Bronchial adenoma.

Fig. 7.—Mural adenoma, right main bronchus. Wedge excision and transverse suture. (Case 8.)

Fig. 8.—Intrabronchial adenoma, left lower lobe bronchus. Left lower lobectomy and excision of adenoma, preserving a flap of bronchial wall for reconstruction. (Case 9.)

illustrates the preservation of an entire right lung which was healthy in spite of an adenoma in the medial wall of the right main bronchus. Wedge resection and transverse suture resulted in complete excision of the tumor with restoration of bronchial continuity.

Bronchial adenomas may be so situated that a tumor, in addition to producing suppuration in the lobe distal to it, has grown so close to another bronchial orifice as to indicate a wider bronchial resection to effect a cure. In such cases it is unnecessary to sacrifice the healthy lung tissue because a bronchoplastic procedure or bronchial anastomosis may be carried out which will permit wide excision of the tumor and yet preserve the healthy lobe. By means of a flap of bronchial wall, it was possible to preserve the left upper lobe in Case 9. Pneumonectomy and loss of function would otherwise have been necessary for adequate removal of the adenoma.

BRONCHOGENIC CARCINOMA

The authors are of the opinion that bronchial reconstructive procedures have a definite place in the surgery of bronchogenic carcinoma under certain limited conditions. By means of transverse suture or dermal grafts, it is possible to restore bronchial continuity following resection of an entire lung, the carina, and a portion of the opposite main bronchus for a bronchogenic carcinoma. This procedure, as suggested by Abbott,[7] extends the operability of certain lesions which impinge upon the carina and trachea as illustrated in Case 10. In certain other instances pulmonary function may be diminished to the degree that it prohibits the resection of an entire lung but will permit the resection of a lobe. By means of a limited resection and bronchial anastomosis, the removal of a carcinoma involving the right main bronchus was made possible in Case 15. Pneumonectomy was out of the question because a contralateral thoracoplasty had been done seven years before for tuberculosis. In two other cases (Cases 11 and 12), lobectomy with wedge resection and bronchial anastomosis were indicated because of poor pulmonary function. In three patients (Cases 13, 14, and 16), a lobectomy and bronchial anastomosis were done deliberately on the premise that these procedures were adequate operations for these particular carcinomas.

Of the seven patients in whom a bronchial reconstruction was done following resection for bronchogenic carcinoma, a total of 8 lobes of lung were preserved. Three patients have died of carcinoma, 4 months, 5 months, and 1 year, respectively, after the operation. It is interesting that in two of these three patients a limited resection and bronchial anastomosis were done as a compromise with pneumonectomy because of poor pulmonary function. The remaining four patients are alive and well, 1, 11, 12, and 18 months, respectively, following the operation. It is noteworthy that three of these four surviving patients were those in whom limited resections and bronchial anastomoses were done deliberately as an adequate operation for their respective carcinomas.

An empyema developed in two patients following transverse suture. Both responded promptly to treatment. A mediastinal and subcutaneous emphy-

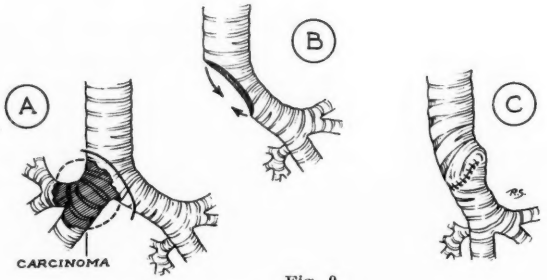


Fig. 9.

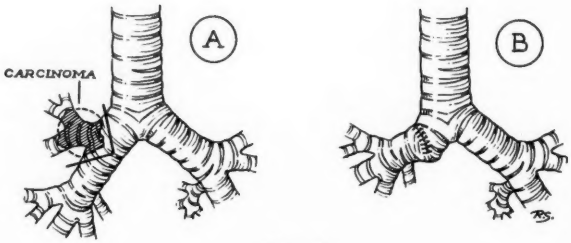


Fig. 10.

Figs. 9-12.—Bronchogenic carcinoma.

Fig. 9.—Epidermoid carcinoma of right main bronchus, trachea, and carina. Resection of right lung, lower trachea, carina, and medial wall of left main bronchus. Reconstruction by transverse suture. (Case 10.)

Fig. 10.—Epidermoid carcinoma, right upper lobe bronchus. Right upper lobectomy, wedge resection of right main bronchus and transverse suture. (Case 11.)

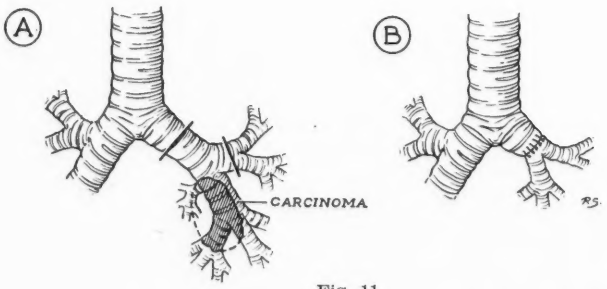


Fig. 11.

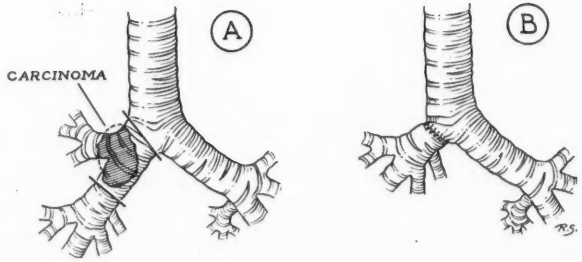


Fig. 12.

Fig. 11.—Carcinoma of left lower lobe bronchus. Resection of left lower lobe and left main bronchus; end-to-end anastomosis of left upper lobe and left main bronchi. (Cases 12, 13, and 14.)

Fig. 12.—Carcinoma of right upper lobe and right main bronchi. Resection of right main bronchus and right upper lobe; end-to-end anastomosis of intermediary bronchus to trachea. (Case 15.)

TABLE IV. BRONCHOGENIC CARCINOMA

CASE	SEX AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
10. L. McC.	M 57	Bronchogenic carcinoma, epidermoid, right main bronchus, trachea, and carina, with lymph node metastases	Resection of right lung, lower trachea, carina, and medial wall of left main bronchus. Reconstruction of left main bronchus by transverse suture of left main bronchus to trachea (Fig. 9)	June 1, 1951	A one year survival. Developed empyema and fistula at site of suture line which healed with drug therapy and drainage. Died June 14, 1952, of carcinoma
11. V. S.	M 56	Bronchogenic carcinoma, epidermoid, right upper lobe bronchus with lymph node metastases. Emphysema with poor respiratory function	Right upper lobectomy, wedge resection of right main bronchus and transverse suture (Fig. 10)	Nov. 11, 1952	Leak at suture line and empyema. Healed with drainage by February, 1953. Well to date
12. S. S.	M 58	Bronchogenic carcinoma, epidermoid, left lower lobe bronchus with lymph node metastases. Emphysema with poor respiratory function	Resection left lower lobe and left main bronchus. End-to-end anastomosis left upper lobe and left main bronchus (Fig. 11)	April 9, 1953	Bronchus healed and left upper lobe expanded. Pelvic metastasis. Died Aug. 30, 1953, with recurrence of carcinoma in bronchus and mediastinum
13. H. E.	M 47	Bronchogenic carcinoma, undifferentiated, superior segmental bronchus, left lower lobe, with lymph node metastases	Resection of left lower lobe and portion of left main bronchus. End-to-end anastomosis, left upper lobe and left main bronchi (Fig. 11)	April 21, 1953	Bronchus healed. Left upper lobe well expanded. Well to date
14. A. B.	M 50	Bronchogenic carcinoma, epidermoid, superior segmental bronchus, left lower lobe, with metastasis to one lymph node	Resection of left lower lobe and portion of left main bronchus. End-to-end anastomosis of left upper lobe and left main bronchi. Tracheotomy (Fig. 11)	June 13, 1953	Emphysema relieved by tracheotomy. Bronchus healed. Left upper lobe well expanded. Well to date
15. C. F.	M 50	Bronchogenic carcinoma, epidermoid, right main bronchus, with metastases to regional lymph nodes. Left 8 rib thoracoplasty for tuberculosis, 1946	Resection right main bronchus and right upper lobe. End-to-end anastomosis of intermediary bronchus to trachea. Tracheotomy (Fig. 12)	June 8, 1953	Bronchus healed. Right middle and lower lobes expanded well. Died Oct. 16, 1953, of carcinoma in right bronchial tree
16. J. K.	M 55	Bronchogenic carcinoma, epidermoid, apical posterior bronchus, left upper lobe. No node involvement	Left upper lobectomy. Left lower lobe bronchus compromised by closure. Wedge resection and transverse closure	March 12, 1954 March 16, 1954	Bronchus healed. Left lower lobe well expanded. Well to date

sema developed immediately postoperatively in one patient (Case 14) due to a leak of air from the bronchial anastomosis and was relieved by tracheotomy. There were no operative mortalities. Recurrence of the carcinoma occurred in the reconstructed bronchial lumen in two patients (Cases 12 and 15). In each instance a localized resection and bronchial anastomosis were done as a compromise with pneumonectomy because of poor respiratory function.

TECHNIQUE

The amazing healing powers of the bronchus have been well demonstrated for years by the simple closures of bronchial stumps used by thoracic surgeons. Experimental and clinical observations with bronchial anastomoses have further substantiated this fact.

No attempts were made to preserve the bronchial vessels in this series of cases. The bronchus was cleanly divided and any bleeding from the bronchial vessels controlled. Careful suturing was done, using interrupted fine cotton or cable wire sutures so placed as to bring the mucosal edges into careful opposition. In so far as possible, the suture was placed through the bronchial wall and submucosa to avoid piercing the mucosa and was tied on the outside of the bronchus. Care was taken to make the closure of the bronchial wall airtight.

Tracheotomy was not done routinely. The bronchial secretions, which were usually troublesome during the first few days postoperatively, were removed in most instances by means of intratracheal catheter and suction. In those instances in which pulmonary function is reduced to an extreme degree, a tracheotomy should be done at the time of the operation, as in Case 15. Should an air leak at the site of the suture line develop in the immediate postoperative period, a tracheotomy should be done to reduce the intratracheal and intrabronchial pressure.

Postoperative bronchoscopy was avoided in most instances. Such instrumentation early in the postoperative period may traumatize the delicate mucosa at the line of suture or reconstruction. Late bronchoscopy for the purpose of inspection of the anastomotic site is unnecessary unless the patient is having symptoms, such as an irritative cough, indicating sutures lying within the lumen of the bronchus. Should this occur, bronchoscopic removal is indicated.

The use of the prone position during any reconstructive procedure involving the bronchus or trachea facilitates the operation considerably from the standpoint of anatomic exposure, anesthesia, and maintenance of a clear airway. Because the main bronchi are situated posteriorly, the most direct exposure for most bronchial anastomoses is obtained with the patient lying in the prone position. In this position the patient tolerates an open bronchus for longer periods of time than in the lateral position because of the absence of the need for positive pressure anesthesia. Indeed, the patient may respire room air through the bronchotomy opening for short periods of time with no deleterious effects.

The airway remains clear of bloody drainage because any bleeding usually drains out into the pleural cavity rather than into the bronchus.

The passage of the intratracheal tube into the contralateral bronchus is usually unnecessary in the prone position. In the case of extensive resections about the carina, however, this is desirable. In some instances in this series of patients, the main bronchus was temporarily closed by means of a soft

clamp, a sponge, or interrupted fine sutures. In one instance (Case 15), a small intratracheal tube was introduced into the distal intermediary bronchus, brought out through the wound, and the two remaining lobes respired by means of a second anesthetic machine.

In the case of bronchogenic carcinoma, the prone position makes possible an extensive mediastinal and pericardial dissection even in those instances in which a lobectomy is done. All lymph-node-bearing areas can be readily approached.

DISCUSSION

There can be no question of the usefulness of bronchial suture, bronchial anastomosis, and bronchoplastic procedures for traumatic bronchial rupture, tuberculous bronchostenosis, and bronchial adenoma. These procedures should be considered in the surgical attack on any benign bronchial lesion to avoid unnecessary sacrifice of good lung tissue.

The utilization of these procedures in the surgical attack on bronchogenic carcinoma must be carefully considered. Inadequate pulmonary reserve and the extension of operability justify their use in certain instances. The authors are of the opinion that, in the interest of preservation of lung tissue and a lower mortality, the operation of lobectomy with wide resection of the carcinoma, detailed dissection of the mediastinum, and bronchial anastomosis may be done deliberately in those cases in which it is believed to be an adequate operation for the tumor.

Our experience with the treatment of bronchogenic carcinoma has indicated that lobectomy, where feasible, yields at least as good results as pneumonectomy, with a lower mortality. The mortality rate in our hands for the 239 resections done for bronchogenic carcinoma during the period from 1945 to 1954 was 10.6 per cent for pneumonectomy and 3.1 per cent for lobectomy (Table V). The survival rates for four years or more for resections done for bronchogenic carcinoma in the years 1945 through 1950 are 25 per cent for lobectomy compared with 13 per cent for pneumonectomy (Table VI). In many instances more favorable lesions, such as the well-localized peripheral

TABLE V. OPERATIVE MORTALITY FOR BRONCHOGENIC CARCINOMA, 1945-1954

TYPE OF RESECTION	NUMBER	DEATHS	MORTALITY (%)
Pneumonectomy	141	15	10.6
Lobectomy	98	3	3.1
Total	239	18	7.5

TABLE VI. SURVIVAL RATES OF PATIENTS WITH BRONCHOGENIC CARCINOMA OPERATED UPON, 1945-1950

TYPE OF RESECTION	NUMBER	SURVIVORS	SURVIVAL RATE (%)
Pneumonectomy	83	11	13.2
Lobectomy	40	10	25.0
Total	123	21	17.0

carcinoma, were removed by lobectomy; although this was not entirely true, the indications being in some instances chest wall involvement and poor pulmonary or cardiac reserve.

Pneumonectomy is the ideal cancer operation in the majority of bronchogenic carcinomas. There remains a group of localized lesions, however, for which lobectomy may be a desirable and practical operation because of the lower mortality rate and the advantages resulting from the preservation of lung tissue. In addition to the indications for lobectomy already given, this operation combined with bronchial resection and anastomosis may be considered for the well-localized, small, centrally located lesions. Furthermore, this procedure will permit wider excision of the bronchus in those patients in whom limited cardiac or respiratory reserve makes lobectomy imperative.

SUMMARY

1. Sixteen cases involving the use of bronchial anastomosis and bronchoplastic procedures in the interest of preservation of lung tissue are presented.

2. These procedures were used successfully for traumatic bronchial rupture (4), tuberculous bronchostenosis (3), and bronchial adenoma (2).

3. Bronchial anastomoses have been utilized for bronchogenic carcinoma in seven patients, because of inadequate pulmonary reserve (3), deliberately for small, centrally located lesions (3), and to extend operability in one patient. Three patients died of their carcinoma within one year after operation. The remaining four patients, in three of whom the operation was done deliberately, are alive after 1, 11, 12, and 18 months, respectively.

REFERENCES

1. Gebauer, P. W.: Plastic Reconstruction of Tuberculous Bronchostenosis With Dermal Grafts, J. THORACIC SURG. **19:** 604, 1950.
2. Gebauer, P. W.: Reconstructive Surgery of the Trachea and Bronchi: Late Results With Dermal Graft, J. THORACIC SURG. **22:** 568, 1951.
3. Gebauer, P. W.: Bronchial Resection and Anastomosis, J. THORACIC SURG. **26:** 241, 1953.
4. Paulson, D. L.: Traumatic Bronchial Rupture With Plastic Repair, J. THORACIC SURG. **22:** 636, 1951.
5. Paulson, D. L.: Plastic Reconstruction of the Trachea and Bronchi, Am. Surg. **18:** 403, 1952.
6. Samson, P., and Evans, B. H.: Personal communication.
7. Abbott, D. A.: Experiences With Surgical Resection of Human Carina, Tracheal Wall, and Contralateral Bronchial Wall in Cases of Right Total Pneumonectomy, J. THORACIC SURG. **19:** 906, 1950.

ADDENDUM

A total of twelve bronchoplastic procedures with resection for bronchogenic carcinoma have now been performed by the authors. The indications were inadequate pulmonary reserve in four, deliberately for centrally located lesions in six, and to extend operability in two patients. One operative mortality occurred twelve days after an end-to-end anastomosis of the left main bronchus and trachea following resection of the right lung and carina. Death was due to inadequate pulmonary function. Three patients died of carcinoma within one year of the operation. The remaining eight patients are alive and well for varying periods up to twenty-six months following operation.

Discussion

(PAPERS BY ABBOTT, VANFLEIT, AND ROBERTO; AND PAULSON AND SHAW)

DR. B. H. EVANS, Fresno, Calif.—I would like to refer briefly to the patient mentioned by Dr. Paulson on whom Dr. Samson and I operated almost a year ago.

(Slide) This early film dated February, 1948, shows a bronchogram with occlusion of the right stem bronchus. This 14-year-old boy at the age of 1 year was run over by a wagon. The accident was followed by a stormy course and hospitalization for three or four months.

(Slide) This is a film made approximately seven weeks after bronchial restoration of the right lung. This shows peripheral filling of the right lung, and I do not believe there is any significant bronchiectasis.

At the time of operation there was mucinous material filling almost the entire left lung, which had to be aspirated from the bronchial tree; the cultures from this secretion were entirely negative.

(Slide) This was made two months after operation. The right lung is well aerated; the diaphragm is elevated, is attached to the chest wall, and is movable on fluoroscopy.

(Slide) This is the most recent slide we have available. The right lung appears to be larger at this time. The trachea is now in the midline, and we hope that the lung will show further expansion.

DR. WATTS R. WEBB, Sanatorium, Miss.—I want to emphasize the point that Dr. Paulson has made, that these lungs that are atelectatic distal to a complete benign stricture do not need to be resected. Dr. Evans and Dr. Samson have demonstrated clinically that they can be re-expanded after a prolonged period of atelectasis. Dr. Burford and I became interested in this some years ago and, in reviewing the pathologic slides of four lungs that had been resected at Barnes Hospital, after as long as twenty-six years of total atelectasis, we found, surprisingly, that they looked almost normal except, of course, for the atelectasis. Experimentally we produced this in dogs by transecting the main stem bronchus, closing both ends, and leaving the lungs atelectatic for periods up to nine months. At the end of that time we reoperated, aspirated all the clear glairy mucus that has been described, and reanastomosed the bronchus. Each lung re-expanded promptly and completely. At a later date we were able to demonstrate a completely normal tracheobronchial tree throughout; we were able to demonstrate a perfectly normal vascular bed and also, by oxygen studies on the blood from both pulmonary veins, to demonstrate that these lungs do oxygenate the blood that is flowing through them. These results were published in the June, 1953, issue of the American Medical Association *Archives of Surgery*, and we were able to predict with confidence that such would be feasible clinically, though we ourselves had not seen such a patient since the development of this concept.

I might make one suggestion on the case that has been presented by Dr. Evans and Dr. Samson; I think that this is not so much a case of incomplete re-expansion of the lung, as that, in the child, the lung did not grow as would the normal lung during the period while it was atelectatic.

DR. J. V. THOMPSON, Indianapolis, Ind.—I certainly appreciated the papers presented by Dr. Abbott and Dr. Paulson emphasizing the applications of these bronchoplastic procedures. My concern is with the application of these procedures to traumatic cases.

(Slide) This case was that of a 33-year-old man who was admitted with a traumatic injury—a crushing injury to the right chest with multiple fractures of the ribs and a tension pneumothorax. The patient was treated at first by a closed thoracotomy with tube drainage. After twelve hours, however, a rapid exudation of air occurred through the catheter, and his condition deteriorated. He was taken to the operating room and explored at once. There was found to be a complete severance of the right upper lobe bronchus and multiple lacerations of the right upper lobe necessitating lobectomy. Lacerations of the superior portion of the right lower lobe were repaired, and that lobe was saved. There was, in addition, however, a long longitudinal rupture of the trachea at the junction of the membranous and cartilaginous

areas, extending superiorly from the carina for 8 cm., then running transversely across the trachea and two cartilages. Oxygenation was maintained by a catheter in the left bronchus, and by use of the balloon on the catheter it was possible to keep the ruptured trachea closed until repair could be accomplished. This was done by approximating sutures after débridement. The patient was followed bronchoscopically, and there has been no evidence of stenosis.

The second case does not demonstrate an occluded bronchus for fifteen years as does the case of Dr. Samson, but the severed bronchus was present for two months before successful repair.

(Slide) The patient was admitted with a traumatic injury and was treated by a closed tube thoracotomy. The condition was complicated by severe cerebral injuries which necessitated waiting for eight weeks before a thoracotomy could be performed. A bronchoscopy was done shortly after admission, and the left main bronchus was found to be occluded by a pulsating mass. Fortunately, the pleural space was filled by the atelectatic lung. At the time of exploration it was noted that the left main bronchus was completely severed. The ends of the bronchus were separated by the pulmonary artery. The ends were dissected free, trimmed, and reanastomosed.

(Slide) Good function of the lung was obtained, and no bronchial stenosis or other complication has occurred in the past year.

DR. ROBERT FOX, Evanston, Ill.—I want to mention a traumatic case similar to that discussed. This one, unfortunately, was at a much higher level. A young boy was admitted to the hospital as an emergency following an automobile accident, with massive bleeding around the face and mouth, and obviously a compound fracture of the mandible. The resident noticed increasing respiratory difficulty, and the patient's neck, which was not lacerated, was starting to puff up and down with each respiration. After controlling the bleeding, the resident started to do a tracheotomy, made an incision, put a finger in to find the trachea, but it was not there. It had retracted about 5 cm. down ino the mediastinum. Fortunately, the boy had been kept face down to evacuate the blood from the pharynx. The resident was able to get a long tracheotomy tube in place while other resuscitative measures were done. The patient was then taken to the operating room and, with a few drops of local anesthesia, the incision was widened and the distal end of the trachea was brought up and found to be ruptured completely at the level of the second tracheal cartilage. There was also a vertical laceration in the membranous portion for about 1.5 cm. downward. This was repaired and then a simple anastomosis was made of the tracheal stumps. Prior to this a new tracheotomy was made through about the fourth and fifth rings, a formal tracheostomy done, and the anastomosis completed. The recurrent laryngeal nerves were both completely ruptured, although the esophagus was not damaged. Of course, he had bilateral cord paralysis but at the present time he is back in school. He has a hoarse voice and some slight dyspnea on exertion.

(Slide) At this level there appears narrowing of the tracheal lumen. The lateral laminogram shows only slight narrowing. He does have a partial stricture and cannot take part in athletics, but he is able to handle himself well. We had not thought of doing anything about the stricture because of the bilateral cord paralysis. The tracheostomy was taken out about two weeks after the anastomosis.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—These two papers have emphasized that the preservation and restoration of function are certainly very fundamental concepts in pulmonary surgery. Probably the enthusiasm with which these concepts are embraced will vary directly with the conscientiousness of the surgeon. I would like to show two cases of bronchial anastomosis that may be instructive—our initial case and our most recent one.

(Slide) The first case is that of an 18-year-old man who presented himself with an empyema on the right. Following drainage, we noticed the atelectasis of the right upper lobe and demonstrated in this bronchogram complete block of the right upper lobe bronchus. Bronchoscopy revealed an adenoma completely blocking this bronchus and extending into the lumen of the main bronchus.

At operation, in July, 1950, we were reluctant to sacrifice the entire lung when we found that the adenoma had directly extended well into the main bronchus itself. The middle and lower lobes were anastomosed directly to the trachea after resecting the major portion of the right main bronchus with the right upper lobe. The next slide shows the final result several months later, with the right middle lobe and right lower lobe satisfactorily filling the right hemithorax.

(Slide) The next case is that of severe tuberculous stenosis in a nurse. The bronchogram demonstrates the marked stenosis of the left main bronchus, with complete block of the left upper lobe.

(Slide) Here is the postoperative film after resection of the destroyed left upper lobe and the stenotic main bronchus. The left lower lobe was anastomosed to the trachea.

I show this next slide to emphasize the importance of postoperative care. Three days after surgery the breath sounds were poor on the left, and obvious block is apparent on this x-ray. Bronchoscopy showed marked reduction of the lumen at the site of the anastomosis due to the temporary edema that always occurs. The final slide shows the complete aeration of the left lower lobe following relief of the temporary obstruction.

The recent article by Gravel in the JOURNAL brought out an important point, I think, in reporting the rupture of an anastomosed bronchus directly into the overlying artery, with fatal termination. We have seen similar rupture of a lobar bronchus into the artery.

To guard against this, we wrap a piece of pleura completely around the anastomosed bronchus in the same fashion as is illustrated in this slide showing our bronchial closure. This interposes an appreciable tissue barrier between the bronchus and the overlying artery, affording considerable protection, in our opinion.

DR. CLIFFORD F. STOREY, Portsmouth, Va.—I should like to compliment both Dr. Abbott and Dr. Paulson on their excellent presentations. My purpose in discussing Dr. Paulson's paper is not to record my triumphs, but to present two cases, one of which developed an unexpected and distressing complication. I would like to ask Dr. Paulson if he knows why this complication occurred and what could have been done to prevent it.

(Slide) This patient had a bronchial adenoma which proved to be of the carcinoid type. It involved the orifice to the middle lobe, the superior segment of the lower lobe, and the main right lower lobe bronchus. I believe that these diagrammatic sketches show the location of the lesion clearly and also explain adequately the type of operative procedure carried out. The lower lobe was resected as was the proximal portion of the middle lobe bronchus and the distal portion of the intermediate bronchus. The stump of the middle lobe bronchus was anastomosed to the stump of the bronchus intermedius just below the right upper lobe bronchial orifice.

(Slide) The film on the left is the preoperative one; the one on the right is a postoperative bronchogram. It is difficult if not impossible to see where the suture line is, and on postoperative bronchoscopy the lumen was entirely adequate.

(Slide) This patient was a comparatively young sailor who also had a bronchial adenoma of the cylindroma type somewhat larger than the previous one and situated in almost precisely the same position in the right lower lobe bronchus involving the margin of the superior segment and the orifice to the middle lobe, necessitating the same type of resection and anastomosis as that carried out in the previous patient.

(Slide) The preoperative chest roentgenogram is shown; he did not have complete bronchial occlusion in spite of the size of the tumor, and actually it is difficult to see in this film that he has a lesion. The x-ray on the right was taken approximately four months after resection and anastomosis, and I must say I was extremely pleased with the appearance of the x-ray. However, I made the mistake of carrying out a postoperative bronchoscopy and, much to my consternation, found that he had developed quite a stricture at the site of the anastomosis. He was kept in the hospital for several additional weeks and the stricture became progressively tighter so that it was necessary to go in and remove the middle lobe which we had attempted to save.

DR. OTTO C. BRANTIGAN, Baltimore, Md.—I wish to compliment Dr. Abbott and Dr. Paulson on the excellent work they have so beautifully presented. I would like to call attention to another type of graft which can be used to restore the trachea and bronchus, a viable pedicle graft using the chest wall; that is, the pleura, periosteum, intercostal muscles, intercostal artery, nerve and vein. I have found little chance to apply this type of graft since the original presentation by Dr. Penton and me. We showed a successful reconstruction of the lower end of the trachea and both main bronchi. There have been two additional patients. One was a 30-year-old woman with a bronchial adenoma involving the right main sten bronchus at the junction of the upper lobe. It was a question of either resecting the whole lung or doing some type of bronchial repair. The adenoma was locally resected, taking out about 3 cm. of bronchus in length and about half of its circumference. The patient has remained well for two years.

The second patient was a 50-year-old man with a contracting localized carcinoma, strangely enough, arising from the upper lobe bronchus, but contracting and invading the main bronchus, the lower end of the trachea, and the left main bronchus. We were able to resect the right lung, the lower end of the trachea, and part of the left main bronchus, reconstructing it with this particular type of viable pedicle graft. The patient did well for three days, and then began to have trouble. He died at the end of four days. The autopsy proved the justification for the operation; all carcinoma had been eradicated locally; there was no lymph node involvement and no distant metastasis. The repair of the lower end of the trachea and left main bronchus was entirely intact, and viable, and certainly would have remained so. The patient died of a massive coronary occlusion.

DR. DEWITT C. DAUGHTRY, Miami, Fla.—I wish to congratulate the authors of the last two papers, for they have presented subjects which are of considerable interest and value. I should like to present four cases that fall into the same general category as presented in both these papers.

The first was a patient, aged 54, with a history of wheezing and slight hemoptysis for one year. He had a negative x-ray of the chest for the entire year, and thus nothing further was recommended in the way of investigation or treatment. At bronchoscopy he was found to have a cylindroma almost completely obstructing the mid and lower trachea. Through a right posterolateral approach, 7 cm. of the lower trachea and posterior carina were excised. Small endotracheal tubes were placed in both stem bronchi and, as the repair was being completed, this was replaced by a larger single plastic tube. The repair was accomplished by the use of tantalum mesh covered with fascia lata. A tube splint was left in the trachea, much like the one presented in Dr. Abbott's paper. This was removed after six days, and the patient's postoperative course seemed quite satisfactory until the eighth day. He developed a tension pneumothorax and a right empyema. An attempt was made to decompress the pneumothorax but was unsuccessful. The patient was in a rather debilitated condition and died with suppuration on the tenth postoperative day.

(Slide) This shows the specimen, 7 cm. in length. The cut section is approximately 2 cm. in thickness.

(Slide) This shows the tantalum mesh and fascia lata repair and reveals that most of the fascia lata has disappeared, thus explaining the tension pneumothorax which was impossible to cope with.

(Slide) Here is the larynx, repaired trachea, and stem bronchi. One can readily see the extensive defect and that the posterior portion of the carina was resected.

Had the plastic tube splint been left in place longer, the outcome might have been different, for it is not unlikely that the lung would have become adherent to the tantalum mesh, thus sealing and making it airtight. The defect was so extensive in this individual that I doubt that a reinforced skin graft would have been successful. The use of intercostal bundles and the attached periosteum might have been a better choice in making the repair.

The three other cases were traumatic. One was a young Negro who had been involved in a motorcycle accident, sustaining a badly fractured larynx and upper trachea. The vocal cords and the adjacent mucosa had been almost completely detached. The larynx was rather badly fragmented and likewise the upper trachea was fractured and badly contused. The vocal cords and mucous membrane were reattached to the laryngeal cartilages and to the trachea. The fracture sites were débrided and closed in a rather meticulous manner. A tracheotomy was not performed, but the tray was kept available, and a resident was in almost constant attendance. A Levine tube was placed in the esophagus. The patient made an uneventful recovery including satisfactory restoration of his voice.

The next case was a Negro who had been involved in a battle of razors and had sustained a completely severed trachea just above the sternum. The thyroid gland had been severed and likewise the left internal jugular vein. The recurrent laryngeal nerves were intact. This patient's trachea was repaired with interrupted sutures without the use of a tracheotomy tube. This patient did not develop any subcutaneous emphysema and was discharged from the hospital on the sixth postoperative day completely recovered.

The last case, and the most extensive injury of this type that we have seen, was a 52-year-old man who was involved in an automobile accident. He was admitted to the hospital with a right tension pneumothorax, in profound shock, and exhibiting marked paradox of the upper anterior half of the right thorax. The skin over this area was intact but the ribs and muscle could not be identified. A closed thoracotomy was performed with the institution of strong suction. The tension pneumothorax remained despite strong suction for four or five hours. It was obvious at this time that the patient had a fracture either of the trachea or of a major bronchus. Through a right anterior thoracotomy approach, it was discovered that he had a fracture of the trachea just above the carina with only 1 cm. of the membranous portion still intact. The fracture extended across the right stem bronchus detaching the upper lobe bronchus. The azygos vein was severed and was actively bleeding into the tracheobronchial tree. The endotracheal catheter was guided into the lung and the left lung was kept inflated while the trachea and right stem bronchus were repaired with interrupted silk sutures. The patient was given the usual postoperative thoracotomy care and made a satisfactory recovery. Three months later he reappeared in the office with a considerable cough, and bronchoscopy revealed a considerable amount of granulation tissue at the site of the suture line in the right stem bronchus and, after removal of some of this granulation tissue, two or three sutures were visible; these were removed and the patient has had no further trouble.

This is certainly a very interesting group of cases. If proper treatment is instituted at an early date, the majority of these people may be salvaged.

DR. E. K. JOHNSON, Brooklyn, N. Y.—I have enjoyed the presentations and the discussion on the various topics. It has resharpened my interest in conserving lung tissue where possible by resecting the bronchial lesion and anastomosing the ends. I know that many of you have been impressed by the rather normal-appearing pulmonary tissue beyond the lesion in the surgical specimen.

I would like to show some cases to illustrate this point. The first is that of a middle-aged man who had a tumor of the right middle lobe. It was diagnosed as an adenoma. At operation it was found that the tumor extended into the right main bronchus, necessitating a bilobectomy, but a sleeve resection of the right main bronchus was done and the two ends anastomosed. On about the eighth day a bronchopleural fistula developed, but was controlled by drainage, and two months later a myoplastic flap occluded the fistula.

The second case was a 34-year-old woman who had an adenoma of the right lower lobe bronchus. Since there was no evidence of suppuration, salvage of the middle lobe was done by resecting the right lower lobe bronchus above the orifice of the right middle lobe, and re-anastomosing it to the right main bronchus.

The third case I shall mention briefly. The patient had a large bulky tumor in the right upper lobe and, in attempting to resect the bronchus at the carina, the opposite main bronchus was partly transected. The partly open end of the trachea was sutured to the cut end of the bronchus. Although I was greatly concerned I found that the closure was exceedingly easy, and the patient had a most unremarkable recovery.

The first case was done two and a half years ago, and a later examination of the specimen showed it to be a carcinoma. He is alive and well today. The second case was done 18 months ago, and the bronchogram shows that the anastomosis is secure and not strictured. The salvaged lobe aerates, does not trap air, and seems to function well. The third case is too recent to comment upon. In each case bronchial secretions were retained for several weeks in the salvaged lobes, and then they finally cleared.

I would like to ask the essayists if they believe that the salvaged lobe is functioning. If secretions are retained, would it suggest that air might be trapped later with emphysema and reduced ventilation? Is it a practical procedure? It appears to me to be practical, but from their greater experience I should like to have their opinion.

DR. GABRIEL P. SELEY, New York, N. Y.—I would like to comment on these two papers with particular reference to adenoma of the bronchus. At Mt. Sinai Hospital we have had an experience with 82 cases over the past twenty years. Two of these were situated at the carina and certainly would be amenable to this type of therapy. However, I would like to caution you on two points. In the first place, many of these adenomas, if not all, have a marked iceberg type formation; that is, most of the lesion is outside the bronchial wall. Therefore, with this type of operative procedure one will probably be able to remove this extrabronchial component with ease. Second, in many cases a lymph node metastasis, particularly by direct extension method, is noted in these adenomas and, in addition (and I do not think this has been stressed), there were four cases with distant metastases. Three of these patients have died of these metastases; two had had previous resections, one a lobectomy and one a pneumonectomy. In the fourth case there were just a few tiny nodules in the liver, but this patient died of amyloidosis twenty-six years after the onset of symptoms. Therefore, for a permanent cure, adequate resection of the extrabronchial component and the regional nodes is necessary for adenoma of the bronchus, even though the malignancy is of very low grade.

DR. LYMAN A. BREWER, III, Los Angeles, Calif.—These papers on tracheal and bronchial reconstruction are extremely important because they place increasing emphasis on preservation of lung tissue. Instead of excising lobes and lungs because of diseased tracheas or bronchi, functioning portions of pulmonary tissue have been saved. The methods that have been so ably described this afternoon should be employed more extensively than is common practice today.

I wish to present briefly one technical point which we have found of great assistance in assuring the success of tracheal and bronchial grafts and anastomoses; that is, the use of a pedicled pericardial fat graft which we presented last year at the San Francisco meeting of this Association. This graft will seal off the leaking of air in the anastomosis better than any other tissue that we have employed. It is a living graft which is adaptable both to bronchial and tracheal anastomosis and reconstruction.

In a case of tracheal resection, performed from a left-sided approach because we thought that it would be necessary to take out the left lung for a tracheal carcinoma, the major portion of the trachea was resected, making an anastomosis up in the neck and below to the carina and to the right main bronchus. Because of the difficult exposure of the right bronchus from the left chest, we had trouble in effecting an airtight anastomosis. The closure, however, was made airtight with the use of a pedicled pericardial fat graft.

Our experience with pericardial fat grafts now totals over 300 cases. The grafts have remained viable for more than three years. As a matter of fact, the blood supply is so good that this method is being used by Dr. Arthur Vineberg to provide additional blood supply to the myocardium. The use of the pedicled pericardial fat graft gives the operator

the assurance that the tracheal or bronchial anastomosis will hold. *We have encountered no early leaks in the first three weeks after operation with this technique.* The late leaks, from three weeks to one or more years, are less than 2 per cent.

DR. JOHN L. POOL, New York, N. Y.—Dr. Paulson has stated that pneumonectomy is the procedure of choice for carcinoma of the lung. I am certainly in agreement with this statement. This procedure, however, is not proper for those patients whose respiratory reserve is too low to survive without being a cripple after the total lung is removed or in cases with other physiologic needs for lesser resection. I should like to call attention briefly to one danger of lesser resections. This is a pathologic point, namely, that whereas the major lymphatic spread from bronchiogenic carcinoma is to the mediastinal lymphatics and also by the veins, one should not forget that there is another rich lymphatic channel in the submucosa of the bronchus and the trachea. It is suggested that where resections of less than total pneumonectomy are contemplated, preliminary bronchoscopic biopsies of the tracheal wall and the major carina proximal to the level of contemplated segmental bronchial resection should be carried out. In several instances after pneumonectomy we have had the misfortune to find at a later date recurrence within the tracheal wall. Also unsuspectedly, on biopsy of the main carina in the study of patients in whom bronchiogenic carcinoma of the main bronchus is present and in whom pneumonectomy is contemplated, cancer has been found in those mucosal lymphatics.

DR. MICHEL LATARJET, Lyon, France.—I appreciate the honor of being a guest of this Association. I have been much interested in the contributions of Dr. Abbott and Dr. Paulson to this subject, and I would emphasize one point. The bronchial reconstruction and almost end-to-end suture of the bronchi which has been traumatized by injury or by carcinoma requires, I think, a very sharp dissection. All around the suture the peribronchial layers are cut off; that means bronchial arteries, some of the bronchial veins to the azygos, pulmonary nerves, and lymph ducts which are going in or coming from the pulmonary tissue. I have been interested in the anatomy of the systemic blood supply to the lung, and I know it has been intensively studied by Dr. Liebow and his associates, but it is still important to emphasize its value.

I had the opportunity of performing such an operation on the bronchi three years ago in a boy, aged 13, after an accident. The main bronchus of the right side was broken and was repaired by end-to-end suture. Now I find that the lung is going very slowly but surely to a bronchiectatic status, and I am afraid the reason is that the bronchial artery was cut around the main bronchus. On endoscopic examination the bronchus is free. There is no stricture, no stenosis. I can only say that when dissecting around the bronchi, one should be careful of the peripheral layers. It is enough to have only 1 cm. of this peribronchial layer, either on the anterior or the posterior aspect of the bronchi, to have a blood supply by the bronchial artery which preserves the lung tissue, which is placed beyond the anastomosis. For there are numerous anastomoses between the bronchial arteries to the lobes; one of the branches of the arterial network, and one branch only, is able to supply the lung.

DR. OSLER ABBOTT, Emory University, Ga. (Closing).—I think this film shows the supporting sutures, the extensive area of resection. Here is a temporary endotracheal tube in place, and here is the prosthesis ready to slip in. While that tube is being placed and adequately fixed, there will be a rather rapid interchange to slip the endotracheal tube within the prosthesis in the distal bronchus area. This stresses the value of the many supporting sutures about the remaining strand of tracheal tissue. Sutures anchoring the prosthesis into the upper end are placed, and after a little hyperventilation we are ready to step from the operating team endotracheal tube over to the anesthetist's tracheal tube which comes down through the larynx. Here is the trade, and with that there is a minimal amount of anoxia. It might be of interest to you to see the balloon expand within the prosthesis.

I merely want to show the extent of resection of the adjacent trachea and bronchus involved in these radical right pneumonectomies, the value of the supporting sutures, and a method for transference of the one tube to the other for use. Now we present a brief demonstration of the size of the dermal graft which has been utilized up to a length of 10 cm. This flap of balsa wood we find helpful in keeping down strain on one's nerves and on one's instruments in placing the pins. A No. 24 silver wire suture is threaded through. It is very helpful to have one man anchor the pin toward which the other man is aiming as the wire is placed. Here the graft is being placed, and it will be sutured into place with multiple interrupted sutures. Over this will be placed a pedicle pleural graft, and particularly with the larger grafts is it mandatory to use the good vascular pedicle graft that Dr. Brewer described. We feel we owe him a great debt for that contribution.

DR. ROBERT R. SHAW, Dallas, Texas.—I want to thank all those who have discussed this paper. Dr. Abbott asked me to answer any questions that might come up with regard to his paper, so that he might show this film. I cannot answer any questions with regard to the grafts he has used. We have used them, but we have not attempted any of the rather extensive resections such as he has shown here. Our main object has usually been to restore the airway by a bronchoplastic procedure rather than the use of a graft.

I would like to comment briefly upon the discussions of Dr. Fox and Dr. Daughtry. One of our residents, approximately three months ago, also encountered a situation where the trachea had been completely torn away from the larynx just below the cricoid cartilage. We have taught our residents and interns in the accident room to do an immediate tracheotomy on patients with crushed chests, especially if there is obvious interstitial emphysema. The intern, in doing the tracheotomy on the man who was just brought in, also could not find the trachea immediately and finally located it with his finger, grasped it with a clamp, pulled it up into the incision, and then called for help. The resident went down to the accident room, and with no more anesthesia than that induced by the already consumed alcohol, was able to do a very nice end-to-end anastomosis, using fine wire, placing the tracheotomy lower down. This man, fortunately, had sustained no injury to the recurrent laryngeal nerves and had a good voice following the repair. I think that is important, because in situations where there has been a traumatic tear or rupture of the trachea, and tracheotomy has been used as an emergency measure without attempting to restore continuity immediately, plastic procedures that may be necessary to restore an airway and eliminate a tracheotomy can be very tedious and usually result in loss of voice.

INTRATHORACIC RUPTURE OF THE TRACHEA AND MAJOR BRONCHI DUE TO CRUSHING INJURY

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THE purpose of this paper is to report a case of rupture of the intrathoracic trachea complicated by avulsion of the right upper lobe bronchus with successful repair and to record a case of complete severance of the left main bronchus satisfactorily anastomosed two months after injury with restoration of pulmonary function.

REVIEW OF THE LITERATURE

A rather extensive review of the literature has failed to reveal a similar case of rupture of the intrathoracic trachea due to a crushing injury of the thorax which has been satisfactorily repaired. The case reported by Khelimskiy[1] may have been similar but the paper was not available for review. Hanner and Cohen[2] successfully repaired a laceration of the intrathoracic trachea, due to a penetrating injury, by the use of a full-thickness skin graft. Henry[3] reported a case of traumatic partial rupture of the lower trachea which was discovered two weeks after injury when granulations obstructed the airway. The patient was treated by bronchoscopic removal of the obstructing tissue.

Zeuch,[4] in 1922, was able to collect 53 cases of rupture of the cervical subcutaneous trachea. Nine of these were treated by tracheotomy but none by open repair. Since then other cases have been reported in the literature.[5-9] Scott described a complete laceration of the trachea and esophagus in the cervical region which was repaired by an end-to-end anastomosis with fine silk. Dysart's[11] patient had a severed trachea near the larynx. A tracheotomy was done and the airway was established by anastomosis over a tube. Nach and Rothman[9] reported 7 cases in each of which there was a lacerated cervical trachea. All of these were repaired by direct suture. A tracheotomy also was performed on two of the patients. An avulsion of the cervical trachea was bridged by a plastic tube through which a tracheostomy tube was inserted by Longmire.[12] Maisel and Dingwall[13] were able to suture a lacerated cervical trachea and establish the airway. Knight[14] treated a stenosis resulting from trauma of the cervical trachea by excision of the scar and replacement by a skin graft with the aid of an intratracheal mold. Fox[15] recently mentioned the successful repair of an injury to the upper cervical trachea and larynx. Daughtry[16] satisfactorily repaired two such injuries of the subcutaneous trachea.

Kinsella and Johnsrud[17] in 1947 were able to find 37 cases of traumatic rupture of the bronchi in the literature. None of these were surgically repaired. Only one-third of these patients apparently survived, although no details were available as to subsequent complications. It is interesting to note that at least

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two patients of this group were able to live over thirty years. These authors reported an additional case which was observed with an atelectatic lung and one case in which a pneumonectomy was performed two years after the injury. Priest[18] described a case in which a thin membrane that was located in the bronchial tree, which resulted from injury, was opened through the bronchoscope to allow aeration of the lung. A similar case was noted in Kinsella's review. Anderson[19] observed a patient with an atelectatic lung seven years after injury, but no surgery was carried out in this case. Santy, Bouchet, and Fraisse[20] reported a case of traumatic injury of the bronchus as did Giovannini.[21] Holinger, Zoss, and Johnston[22] noted a complete occlusion of the bronchus two months after injury in a case in which there was no operative procedure. These authors also described two other patients with rupture of a bronchus. A pneumonectomy was performed upon one five weeks after injury and in the other, three years after injury. Hodes, Johnson, and Atkins[23] performed a pneumonectomy five months after a complete separation of the bronchus. Kirkpatrick[24] performed a pneumonectomy for a completely divided right bronchus immediately after injury.

Scannell[25] was able to repair the lacerated membranous portion of a right main bronchus with fine interrupted silk sutures. Two cases of rupture and two of occluded bronchi were described by Paulson[26] and Shaw[27] in which they utilized bronchoplastic procedures and the dermal graft. Daughtry[16] was able to repair a rupture across the right main bronchus extending into the trachea, but the patient was troubled with granulations forming at the suture line as has been noted by other authors. Samson[28] and his associate recently described the case of a young man with a history of injury fifteen years prior to surgical reconstruction of the bronchus which resulted in an aerated lung.

Griffith[29] repaired a completely severed left main bronchus seven months after injury with an end-to-end anastomosis surrounded by a muscle graft. Weisel and Jake[30] anastomosed a severed right main bronchus directly to the trachea forty-six days after injury.

Experimental work on the repair of the tracheobronchial tree has been carried out by several investigators. Grindlay and Clagett with their colleagues[31, 32] demonstrated the technical difficulties encountered and the tendency to stenosis in using polyethylene tubing to bridge large defects. Craig, Holmes, and Shabart[33] likewise encountered stenosis in the use of a long polythene prosthesis. Bucher, Burnett, and Rosemond[34] utilized stainless steel mesh with some success in cervical tracheal defects but with poor results in the intrathoracic area. Daniel[35] repaired tracheal defects in animals with metal or glass tubes and demonstrated the difficulty of expectorating through prostheses and of maintaining them in place. Fascial grafts covered with surrounding connective tissue in his hands resulted in stenosis. Taffel[36] pointed out the value of fascial grafts to cover windows in the tracheobronchial tree. Jackson and Tuttle with their colleagues[37, 38] stated that fresh homologous tracheal transplants were of little value whereas short preserved homologous grafts were good and acted as bridges for new tissue growth. In their experience long grafts collapsed and

produced stenosis. They further pointed out that crushing of the bronchi caused poor healing of the anastomosis. Their best results were obtained from direct anastomosis after section of the bronchial tubes. Gelfoam was found by them to be a good bridge for small windows in the airways as was noted by Hanlon.[39] Marrangoni[40] found experimentally that homotransplants of lyophilized tracheal tissue were replaced in time with connective tissue which produced a satisfactory bridge for only short defects. Ferguson, Wild, and Wangensteen[41] had poor experimental results with grafts but had good results with resection and end-to-end anastomoses, particularly where the mucosa was accurately approximated. Daniel and associates[42] also had good success from direct anastomosis of the airways and found that results were equally good with the use of homologous or autogenous grafts to repair windows in the tracheobronchial tree. Davies, Edmiston, and McCorkle[43] found that, in animals, preserved homologous grafts produced a good repair of small windows and when utilized over acrylic tubes in large defects, but complete stenosis resulted from the use of these grafts alone in the replacement of large defects of the trachea. Carter and Strieder[44] have made an experimental study on the resection of the trachea and bronchi. Gebauer[45, 46] has adequately shown the value of wire-reinforced dermal grafts for the repair of smaller defects and in the enlargement of stenosed areas in the tracheobronchial tree.

These methods and techniques have been applied clinically to repair defects in the major airways following radical excision for carcinoma or defects produced by the excision of benign neoplasms as well as for the surgical enlargement of fibrous stenoses of the bronchus.[27, 45-55] Penton and Brantigan[56] repaired a large defect in the trachea produced by the excision of a tumor by the ingenious method of utilizing a pedicled graft of the thoracic wall. The flap consisted of intercostal muscles, periosteum and pleura. This tissue was apparently selected in an effort to produce stability of the area by bony regeneration.

CASE REPORTS

CASE 1.—The patient was a white man, 34 years of age, whose right thorax was crushed by a heavy concrete tile. The patient suffered from pain and dyspnea and expectorated bright blood. Cyanosis, tympany, absent breath sounds, subcutaneous emphysema, and bony crepitation were noted over the right thorax. There was a history of a previous gastroenterostomy. Roentgenologic examination disclosed a hydropneumothorax and multiple fractures of the upper five ribs on the right. The patient was given supportive therapy, and an intercostal catheter was inserted in the right pleural cavity for decompression purposes. Considerable relief was obtained by these measures. Early the next morning there was a rapid deterioration of the patient's condition. Severe hemoptysis and increasing dyspnea rapidly occurred. A continuous stream of air issued from the intercostal tube. A marked increase of the pneumothorax with displacement of the mediastinum was noted despite the decompression (Fig. 2). A ruptured major airway was considered to be present.

A thoracotomy was immediately performed upon the right thorax. Prior to induction of anesthesia the blood pressure rose from 110/70 to 210/50 mm. Hg. Compression of the gas bag caused large quantities of gas to issue from the intercostal catheter which had been left in place as a safety factor until the thorax was opened, when air gushed from the incision. A large quantity of blood was evacuated from the pleural cavity. The right upper lobe bronchus was completely avulsed from the trachea and the lobe was so damaged by multiple lacerations that a lobectomy was performed. Oxygenation was maintained by

catheterizing the left bronchus with a long intratracheal tube. The tracheal stump of the bronchus was carefully trimmed of injured tissue and sutured with interrupted 00 silk. An extensive longitudinal rupture of the right tracheal wall was present at the juncture of the membranous and cartilaginous portions extending upward for about 8 cm. from a point 1.5 cm. above the bifurcation of the trachea and was not connected to the site of bronchial injury (Fig. 1, *A*). The superior end of the defect also extended transversely and anteriorly across a fractured cartilage for about 1.5 cm. The azygos vein was divided and ligated. The edges of the torn tracheal walls were trimmed of devitalized tissue and the edges including the mucosa were carefully approximated with interrupted sutures of 00 silk. A flap of pleura was attached over the suture line with 00 catgut. The endotracheal tube was then retracted into the trachea from the left bronchus. Positive pressure was applied which demonstrated that the repair was airtight. The remaining lobes expanded to fill the hemithorax. A laceration of the superior division of the lower lobe was sutured with 00 catgut. Intrapleural protrusions of the fractured ribs which had lacerated the lung were rongeured away and the fractures were immobilized by sutures placed in the adjacent intercostal tissues. Three large catheters were inserted into the pleural cavity through stab wounds for decompression and the incision was closed. The patient's condition improved rapidly through the operative procedure.

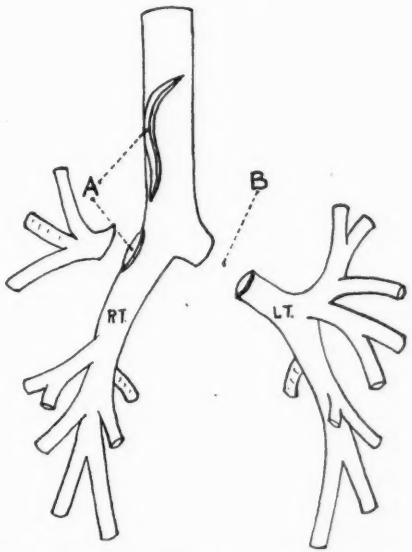

Fig. 1.—*A*, Site of rupture of the intrathoracic trachea and avulsion of the right upper lobe bronchus in Case 1. *B*, Site of severance of the left main bronchus in Case 2.

A soft tube was inserted through the nose into the trachea to separate the vocal cords in order to prevent the development of increased intratracheal pressure and to provide a means of aspirating pulmonary secretions. Periodic instillations of Pontocaine permitted the patient to tolerate the inlying tube. Oxygen was supplied through the tube until it was removed on the fifth day. The patient was able to swallow liquids despite its presence. Massive doses of antibiotics were administered and the blood loss was replaced by transfusion.

The patient's condition rapidly improved during the postoperative period, and he was discharged on the fifteenth postoperative day with the remaining lobes well expanded (Fig. 3).

The patient returned to his occupation and his condition was observed for a year after his injury. Tracheoscopic examinations were made at intervals of approximately three months, and there has been no evidence of stenosis.

This case is, therefore, that of a crushing injury to the chest resulting in rupture of the intrathoracic trachea which was successfully repaired with no development of stenosis. An additional complication of avulsion of the right upper lobe bronchus with severe lacerations of the lobe required lobectomy.

CASE 2.—The patient was a white man, 58 years of age, who was pinned beneath an overturned tractor. He was in a semicomatose condition and a state of shock for which

Fig. 2.—Roentgenogram illustrating the tension pneumothorax with mediastinal displacement despite decompression.

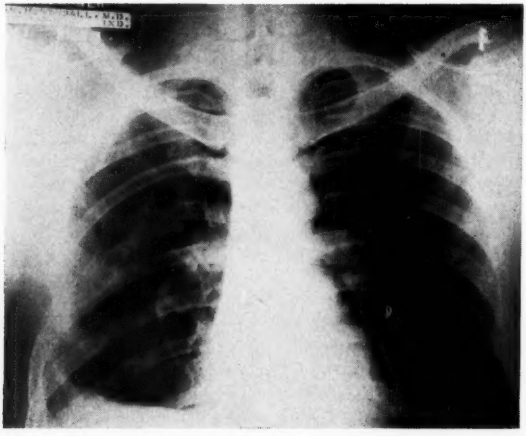

Fig. 3.—Roentgenogram at time of discharge from the hospital following surgical repair of the injured intrathoracic trachea.

supportive therapy was administered. A left pneumothorax was decompressed by intercostal tube drainage. Tracheotomy was performed to maintain patency of the airway because of the presence of cerebral injuries, pulmonary edema, and paradoxical motion of the thoracic wall due to multiple fractures of the ribs. Right hemiplegia and decubitus ulcers developed. The patient remained stuporous and semicomatose for a number of days. Neurosurgical consultation was had with Drs. E. Vernon Hahn and Paul Merrell. An electrocardiogram disclosed evidence of an old posterior coronary occlusion. An atelectatic left lung obliterated the space in the left pleural cavity by the third day, and a bronchoscopic examination revealed an obstruction of the left bronchus by lacerated bronchial tissues (Fig. 4). The nutrition was maintained through a Levine tube and by parenteral administration for the first week. Approximately a month after injury a bronchoscopy was again performed, which disclosed that the left main bronchus was obstructed by a membranous pulsatile mass of tissue. The diagnosis was that of a ruptured bronchus with resultant atelectasis of the left lung.

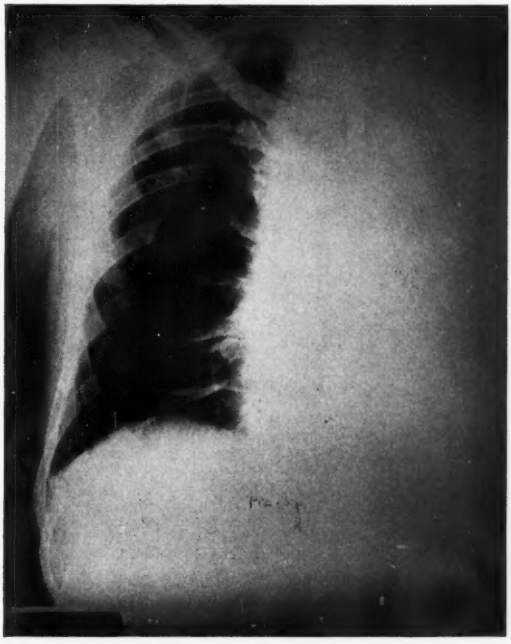

Fig. 4.—Roentgenogram taken three days after injury, demonstrating the atelectatic left lung obliterating the hemithorax. The condition persisted for two months until surgical intervention.

Two months after injury the patient's condition improved sufficiently to permit a thoracotomy. Adhesions between the atelectatic lung and the thoracic wall were separated and a protruding fractured rib was rongeured away. An underlying laceration of the lung was repaired by suture. Dissection of the hilum disclosed a complete division of the left main bronchus with the pulmonary artery interposed between the separated ends of the bronchus (Fig. 1, *B*). The artery was mobilized by a tedious dissection through a mass of fibrous tissue and the bronchial stumps were exposed. The ends of the bronchus were obliterated by firm fibrous membranes. Both stumps were amputated to provide clean undamaged tissue for end-to-end anastomosis inasmuch as two cartilaginous rings on each side had been frac-

tured by the injury. A large quantity of thick tenacious seromucoid material was thoroughly aspirated from the distal stump and lung. A careful end-to-end anastomosis with good approximation of the mucosa was carried out with interrupted sutures of 00 silk, and a small piece of Gelfoam was used to surround the site of the anastomosis for reinforcement. The atelectatic lung became aerated readily after this procedure. The thoracic wall was closed following the insertion of two intercostal tubes into the pleural cavity for decompression.

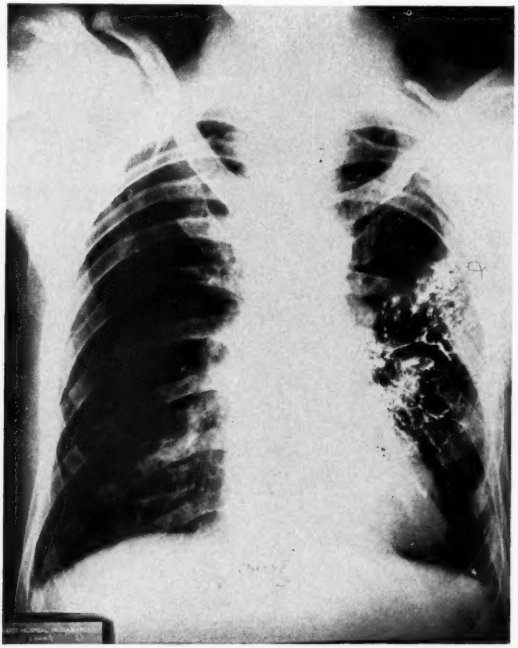

Fig. 5.—Postoperative roentgenogram demonstrating the fully reaerated left lung after anastomosis of the severed main bronchus.

The postoperative course was marked by complete expansion of the lung. Secretions were aspirated through the tracheostomy tube which had been previously placed and was not removed until two weeks after the operation. Antibiotic therapy was maintained and the poor state of nutrition was improved by appropriate measures. A bronchoscopic examination, performed three weeks after the thoracotomy, revealed a widely patent left main bronchus with no tendency to stenosis or evidence of granulation tissue at the suture line. The patient was then discharged from the hospital.

The patient was subjected to bronchoscopic examination at intervals of three months for one year and no evidence of stenosis appeared at any time. Bronchograms disclosed no signs of bronchiectasis (Fig. 5). The lung continued to function well but a residual hemiplegia has prevented the patient from resuming his previous occupation as a farmer.

The second case is thus that of a completely severed left main bronchus with interposition of the pulmonary artery successfully repaired by end-to-end anastomosis two months after crushing injury with restoration of pulmonary function uncomplicated by stenosis or bronchiectasis.

DISCUSSION

Rupture of the major intrathoracic airways, especially of the trachea, from crushing injuries of the thorax must be exceedingly rare as evidenced by the paucity of reports in the literature. Undoubtedly, there will be an increase in this type of injury with modern high-speed transportation. The mechanism of rupture must be related to a shearing force with a sudden increase in the intrathoracic pressure or to a sudden increase in the intratracheal pressure with the glottis tightly closed. On the other hand, rupture of the cervical subcutaneous trachea was reported in 53 cases prior to 1922 and several additional cases have been reported since that date. The subcutaneous trachea also is more exposed to direct injury and the injury should be more readily diagnosed. Direct attempts to repair the trachea, however, were reported in only one-sixth of these cases.

Only 37 cases of ruptured bronchi were reported by 1947, and none of these were subjected to surgical intervention, with the result that two-thirds of these patients died. An occasional patient lived a number of years with an atelectatic lung apparently due to spontaneous healing at the site of injury. The others probably suffered numerous suppurative complications. Twenty additional cases of rupture of the bronchus have been reviewed which were reported since 1947. Five of these were submitted to pneumonectomy and nine to plastic repair of the injured area. In one case a thin membrane formed at the site of injury and continuity of the airway was restored by bronchoscopic means.

If actual defects in the tracheobronchial tree should be created by injury, fascial and dermal grafts appear to offer satisfactory means of replacing the smaller ones. Dermal grafts, however, seem to offer the best means at present for the repair of these small defects and for bronchoplastic procedures on stenosed major airways. Well-fitting autogenous and preserved homologous tracheobronchial grafts apparently may be utilized for the repair of small areas as they act as bridges for tissue replacement. These grafts, however, seem to be less practical for general usage. Various prostheses have not been too successful, particularly when utilized to bridge large defects because of their tendency to migration, interference with drainage, collapse, and stenosis. Fascial grafts reinforced with tantalum or steel mesh probably have value for the replacement of large tracheobronchial defects.

Direct anastomosis and suture repair of the defects where feasible and the use of pedicled flaps offer the best means of reconstructing the tracheobronchial tree at present. Viable, smooth, well-approximating edges are essential to primary union and the prevention of granulations forming at the suture line. Maintenance of an adequate airway to the functioning respiratory area by intubation is essential during the operative procedure. A tracheostomy or an inlying endotracheal tube during the early postoperative period is of value in the prevention of increased intrabronchial or intratracheal pressure with tension on the suture line and in the removing of secretions. An intratracheal tube may be maintained in place by the use of Pontocaine spray to prevent irritation or coughing. In those cases where surgical intervention was not carried out at

the time of injury and the patient survives, fibrous membranes cover the severed ends of the airways. The stumps, therefore, must be amputated and the seromucoid fluid trapped in the lung must be aspirated. The successful reconstruction of the tracheobronchial tree following crushing injury, as noted in the recent literature and as illustrated in the two cases just reported, leaves little place for pulmonary resection unless there is irrecoverable pulmonary damage.

The diagnosis of a ruptured major intrathoracic airway may be based on the presence of a tension pneumothorax with increasing dyspnea despite apparently adequate decompression and the almost continuous flow of air from the intercostal tube. The critical condition of the patient usually demands emergency repair to maintain oxygenation and relieve mediastinal compression. Spontaneous closure of a defect as noted previously may occur from occlusion by the adjacent tissues. In this instance operative intervention is indicated as soon as the patient's condition permits, in an effort to restore pulmonary function and prevent suppuration of the lung in the future. Atelectasis occurring after thoracic injury, particularly with a history of pneumothorax or hemoptysis and bronchoscopic evidence of occlusion, suggests the diagnosis in this circumstance. The plastic reconstruction of previously severed or occluded bronchial tubes apparently may be carried out several months to several years following an old injury with restoration of pulmonary function.

SUMMARY

The pertinent information contained in the literature has been reviewed and discussed. A case of successful repair of a ruptured intrathoracic trachea complicated by an avulsed lobar bronchus and a case of satisfactory delayed repair of a severed main bronchus resulting in a functioning lung following crushing injury to the thorax have been described in some detail. The apparent rarity of these injuries, the diagnosis, management, and salient points in the reconstruction of the tracheobronchial tree have been discussed in the paper.

REFERENCES

1. Khelimskiy, M. A.: Case of Successful Suture of Trachea in Its Lower Segment, Khirurgiya **2**: 84, 1945.
2. Hanner, J. M., and Cohen, S. H.: Repair by Full-Thickness Skin Graft of Penetrating Wounds of the Intrathoracic Trachea, J. THORACIC SURG. **25**: 380, 1953.
3. Henry, G. A.: Traumatic Rupture of the Lower Trachea With Stenosis, Canad. M. A. J. **64**: 134, 1951.
4. Zeuch, L. H.: Subcutaneous Rupture of the Trachea, Ill. Med. J. **41**: 451, 1922.
5. Nixon, J. W.: Spontaneous Subcutaneous Rupture of Trachea, J. A. M. A. **85**: 1221, 1925.
6. Oliva, C.: Case of Rupture of Trachea Following an Effort, Clin. chir. **31**: 19, 1928.
7. Wolff, S.: Subcutaneous Rupture of Trachea, Jahrb. f Kinderh. Berlin, **108**: 57, 1925.
8. Metson, B. F.: Subcutaneous Rupture of the Trachea, A. M. A. Arch. Otolaryng. **57**: 182, 1953.
9. Nach, R. L., and Rothman, M.: Injuries to the Larynx and Trachea, Surg., Gynec. & Obst. **76**: 614, 1943.
10. Scott, G. D.: Extensive Cut-throat With Complete Laceration of Trachea and Esophagus, J. A. M. A. **90**: 689, 1928.
11. Dysart, B. R.: Subcutaneous Rupture of Trachea From Larynx by External Trauma: With Stenosis and Recovery, Laryngoscope **59**: 502, 1949.
12. Longmire, W. P. Jr.: The Repair of Large Defects of the Trachea, Ann. Otol. Rhin. & Laryng. **57**: 875, 1948.
13. Maisel, B., and Dingwall, J. A.: Primary Suture of the Divided Cervical Trachea, Surgery **27**: 726, 1950.

14. Knight, J. S.: Injuries of the Trachea and Esophagus Incurred in Combat, Ann. Otol. Rhin. & Laryng. **55**: 656, 1946.
15. Fox, R.: Discussion of reference 27.
16. Daughtry, D. C.: Discussion of reference 27.
17. Kinsella, T. J., and Johnsrud, L. W.: Traumatic Rupture of the Bronchus, J. THORACIC SURG. **16**: 571, 1947.
18. Priest, R. E.: Unusual Conditions Simulating Pulmonary Atelectasis, Ann. Otol. Rhin. & Laryng. **59**: 889, 1950.
19. Anderson, B. W.: Traumatic Rupture of the Left Main Bronchus, Brit. J. Tuberc. **43**: 40, 1949.
20. Santy, P., Berard, M., Bouchet and Fraisse, P.: Pulmonary Suppuration Due to Traumatic Rupture of the Left Bronchial Trunk, Lyon Chir. **40**: 672, 1945.
21. Giovannini, S.: Traumatic Laceration of a Large Right Bronchus With Mediastinal Emphysema, Minerva Chir. **4**: 400, 1949.
22. Holinger, P. H., Zoss, A. R., and Johnston, K. C.: Rupture of Bronchus Due to External Chest Trauma; Report of 3 Cases With Recovery, Laryngoscope **58**: 817, 1948.
23. Hodes, P. J., Johnson, J., and Atkins, J. P., Traumatic Bronchial Rupture With Occlusion, Am. J. Roentgenol. **60**: 448, 1948.
24. Kirkpatrick, E. R. G.: Case of Traumatic Avulsion of Main-stem Bronchus From Its Lung, Treated by Immediate Pneumonectomy, Brit. J. Surg. **37**: 362, 1950.
25. Scannell, J. G.: Rupture of Bronchus Following Closed Injury to Chest; Report of Case Treated by Immediate Thoracotomy and Repair, Ann. Surg. **133**: 127, 1951.
26. Paulson, D. L.: Traumatic Bronchial Rupture With Plastic Repair, J. THORACIC SURG. **22**: 636, 1951.
27. Paulson, D. L., and Shaw, R. R.: Bronchial Anastomosis and Bronchoplastic Procedures in the Interest of Preservation of Lung Tissue, Presented at the Meeting of The American Association for Thoracic Surgery, Montreal, May 5, 1954.
28. Samson, P.: Discussion of reference 27.
29. Griffith, J. L.: Traumatic Fracture of the Left Main Bronchus, Thorax **4**: 105, 1949.
30. Weisel, W., and Jake, R. J.: Anastomosis of the Right Bronchus to the Trachea Forty-six Days Following Complete Rupture From External Injury, Ann. Surg. **137**: 220, 1953.
31. Grindlay, J. H., Clagett, O. T., and Moersch, H. J.: Resection and Anastomosis of Trachea; An Experimental Study, Ann. Otol. Rhin. & Laryng. **58**: 1225, 1949.
32. Swift, E. A., Grindlay, J. H., and Clagett, O. T.: The Repair of Tracheal Defects With Fascia and Tantalum Mesh; An Experimental Study, J. THORACIC SURG. **24**: 482, 1952.
33. Craig, R. L., Holmes, G. W., and Shabart, E. J.: Tracheal Resection and Replacement With a Prosthesis, J. THORACIC SURG. **25**: 384, 1953.
34. Bucher, R. M., Burnett, W. E., and Rosemond, G. P.: Experimental Reconstruction of Tracheal and Bronchial Defects With Stainless Steel Wire Mesh, J. THORACIC SURG. **21**: 572, 1951.
35. Daniel, R. A. Jr.: The Regeneration of Defects of the Trachea and Bronchi; An Experimental Study, J. THORACIC SURG. **17**: 335, 1948.
36. Taffel, M.: The Repair of Tracheal and Bronchial Defects With Free Fascia Grafts, Surgery **8**: 56, 1940.
37. Jackson, T. L., Lefkin, P., Tuttle, W., and Hampton, F.: An Experimental Study in Bronchial Anastomosis, J. THORACIC SURG. **18**: 630, 1949.
38. Jackson, T. L., O'Brien, E. J., Tuttle, W., and Meyer, J.: The Experimental Use of Homogenous Tracheal Transplants in the Restoration of Continuity of the Tracheobronchial Tree, J. THORACIC SURG. **20**: 598, 1950.
39. Hanlon, C. R.: Observations on the Use of Gelatin Sponge in Closure of Experimentally Produced Defects of the Bronchus, Surg., Gynec. & Obst. **86**: 551, 1948.
40. Marrangoni, A. G.: Homotransplantation of Tracheal Segments Preserved by Lyophilization; An Experimental Study, J. THORACIC SURG. **22**: 568, 1951.
41. Ferguson, D. J., Wild, J. J., and Wangensteen, O. H.: Experimental Resection of the Trachea, Surgery **28**: 597, 1950.
42. Daniel, R. A., Taliaferro, R. M., and Schaffarzick, W. R.: Experimental Studies on Repair of Wounds and Defects of the Trachea and Bronchi, Dis. Chest **17**: 426, 1950.
43. Davies, O. G., Edmiston, J. M., and McCorkle, H. J.: The Repair of Experimental Tracheal Defects With Fresh and Preserved Homologous Tracheal Grafts, J. THORACIC SURG. **23**: 367, 1952.
44. Carter, M. G., and Strieder, J. W.: Resection of Trachea and Bronchi; An Experimental Study, J. THORACIC SURG. **20**: 613, 1950.
45. Gebauer, P. W.: Further Experiences With Dermal Grafts for Healed Tuberculous Stenosis of Bronchi and Trachea, J. THORACIC SURG. **20**: 628, 1950.
46. Gebauer, P. W.: Reconstructive Surgery of the Trachea and Bronchi: Late Results With Dermal Grafts, J. THORACIC SURG. **22**: 568, 1951.

47. Kergin, F. G.: Carcinoma of the Trachea, J. THORACIC SURG. **23**: 164, 1952.
48. Kay, E. B.: Tracheal Resection With Primary Anastomosis, Ann. Otol. Rhin. & Laryng. **60**: 864, 1951.
49. Abbot, O. A., Van Fleit, W. E., and Roberto, A.: Experiences With Enlarging the Indications for Tracheal and Bronchial Grafts, Presented at the Meeting of The American Association for Thoracic Surgery, Montreal, May 5, 1954.
50. Storey, C. F.: Discussion of reference 49.
51. Johnson, J.: Discussion of reference 49.
52. Cahan, W. G.: Carcinoma of Intrathoracic Trachea: Excision and Repair by Tantalum Gauze—Fascia Lata Graft, J. THORACIC SURG. **23**: 513, 1952.
53. Cotton, B. H., and Penido, Joao, R. F.: Resection of the Trachea for Carcinoma; Report of 2 Cases, J. THORACIC SURG. **24**: 231, 1952.
54. Sealy, W. C., Keeley, R. L., Collins, J. P., and Stephen, C. R.: Reconstruction of Benign Non-Tuberculous Strictures of the Trachea, Ann. Surg. **138**: 99, 1953.
55. Gravel, J. A.: Bronchial Resection and Anastomosis, J. THORACIC SURG. **27**: 244, 1954.
56. Penton, R. S., and Brantigan, O. C.: The Use of a Viable Pedicle Graft for Repairing an Extensive Tracheobronchial Defect, Ann. Surg. **135**: 709, 1952.

OIL GRANULOMA AND LIPOID PNEUMONITIS: A COMPLICATION OF OLEOTHORAX

Report of Seven Cases

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THE use of mineral oil or paraffin as a method of plombage in the chest cavity has largely fallen into disuse in this country, although it has not been entirely discarded, and it is still rather widely used in several foreign countries. Adams[1] only recently reported on the use of paraffin in over 200 cases, and stated that in none was there evidence of migration of the substance. Cutler[2] continues to use oil as a plomb in extrapleural pneumothorax and states that he has encountered no trouble with it. Gaensler and Strieder[3] have recently used oil as plombage in a few selected cases and have observed no ill effects.

In contrast to these favorable reports, however, a survey of the literature will reveal that a number of cases have been reported in which oil granuloma formation or oil pneumonia has occurred. Matson,[4] one of the earlier proponents of the use of oil, reported the formation of paraffinomata in 12 per cent of 50 cases in which oil in the form of Gomenol was used for collapse. Browning, Ray, and Rotenberg[5] reported in 1946 on the late investigation of 101 cases which they had studied six years earlier. Of the 75 patients which they had considered successful, eight had developed bronchopleural fistula, and of these eight, five had died. Oil aspiration presumably was a contributory factor. They found that after six years of oleothorax, there was no case in which re-expansion of the lung occurred when the oil was withdrawn.

Of especial interest are those cases in which mediastinal oil granuloma has occurred with esophageal compression. We have been able to find eight such cases reported in the literature.[6-10]

Ballantyne, Clagett, and McDonald,[6] in addition to reporting a case of esophageal obstruction due to oil granuloma, also reported a case of oil granuloma in the chest wall and breast and another in which granuloma formation around the brachial plexus produced severe brachial plexus neuritis. They quote Livieratoes as having reported cases with migration of oil into the chest wall and breast and to the opposite thorax.

Nagel[11] has also reported a case with contralateral migration and granuloma formation in the pleura and pericardium.

MATERIAL

There have been 76 cases at Nopeming Sanatorium in which mineral oil in the form of Gomenol has been used as a plombage method, either intrapleural or extrapleural. In addition there have been two cases treated by

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extrapleural plombage with paraffin. In seven cases complications have developed which were directly due to the presence of oil or paraffin and the granulomatous reaction which these agents produce. In addition to these seven cases, there were many others in which oil proved to be an ineffective therapeutic agent, and in a considerable number of cases complications due to tuberculosis occurred. However, of primary interest are those seven in which the oil per se caused the trouble.

CASE REPORTS

CASE 1.—P. V., a 68-year-old former iron miner, was admitted to Nopeming Sanatorium Aug. 19, 1940, with bilateral, far-advanced tuberculosis and cavitation in the right upper lobe.

A right artificial pneumothorax was started on Aug. 30, 1940, but was unsuccessful because of the development of tuberculous empyema. Repeated thoracenteses were done on the right but became progressively difficult, and multiple subcutaneous abscesses developed.

On May 25, 1941, the pneumothorax was converted to an oleothorax, which resulted in some improvement. However, in March, 1942, two pleurocutaneous fistulas developed, from which oozed oily pus. These fistulas persisted in spite of many types of treatment.

In August, 1942, the patient had a cerebrovascular accident with a resultant hemiplegia, and his condition steadily declined until Oct. 16, 1942, at which time he died.

Permission for autopsy was refused.

CASE 2.—L. M., a 22-year-old male laborer, was first admitted in October, 1933, with moderately advanced pulmonary tuberculosis, bilateral, and with cavitation in the left lung. Anorectal tuberculosis was also present at this time.

In March, 1934, a fistula in ano was unroofed, and in July, 1936, a right pneumothorax was begun, followed by a left pneumothorax in September, 1936. These measures were ineffective and were abandoned. A right extrapleural pneumonolysis was done in March, 1942, and the resultant pocket was converted to an oleothorax. The same procedures were carried out on the left in March, 1944, resulting in marked improvement, and the patient left the sanatorium against advice in May, 1945.

In September, 1946, he was readmitted with a tuberculous empyema on the right side which was treated by aspirations. A subcutaneous oil abscess developed at the site of the aspirations. This was very difficult to treat. In order to obliterate this area, it ultimately became necessary to do a five-rib thoracoplasty. Caseous necrotic material was found at the site of the abscess in the area of the interspaces. After thoracoplasty the patient improved and was discharged in 1948. To date he has remained well and is working.

CASE 3.—M. S., a 61-year-old Polish housewife, was admitted to Nopeming Sanatorium, April 4, 1940, with far-advanced bilateral pulmonary tuberculosis and cavitation in the right upper lung. She also had diabetes mellitus.

A right pneumothorax was begun in September, 1940, and was carried until December, 1941, when it was abandoned and oleothorax was substituted. This procedure was not effective in converting the patient's sputum but was carried for the next ten years.

In August, 1950, she began to expectorate oil, and immediately thereafter a new mottled type of infiltration showed up in the left lower lobe. The patient died three weeks later.

Autopsy revealed bilateral, active, pulmonary tuberculosis with a large, right-sided cavity and a bronchopleural fistula. An empyema and oleothorax were encountered on the right, and an extensive pneumonitis was found in the left lung.

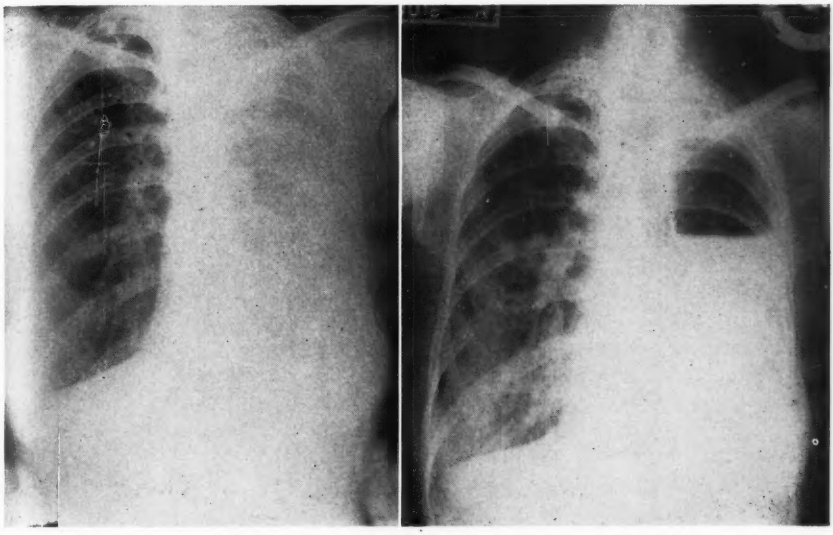
CASE 4.—R. R., a 35-year-old housewife, was admitted to Nopeming in February, 1939, with moderately advanced, bilateral pulmonary tuberculosis and cavitation.

Pneumothorax was begun on the right within a few days after admission, and shortly thereafter an intrapleural pneumonolysis was done on that side. A left pneumothorax was

begun in April, 1939, followed by a left intrapleural pneumonolysis. Because of obliterative pleuritis the right pneumothorax was converted to an oleothorax on Aug. 23, 1943.

In August, 1946, a massive spread of disease in the right lung was apparent. A pneumoperitoneum was begun, but in November, 1946, the patient began expectorating oil, and a bronchopleural fistula became evident. The remainder of the oil was aspirated from the pleural space, but the patient went into acute right heart failure and died. Unfortunately, autopsy permission could not be obtained.

CASE 5.—C. C., a 23-year-old white girl, was admitted to Nopeming Sanatorium in December, 1938, with bilateral, moderately advanced pulmonary tuberculosis and bilateral cavitation. A left pneumothorax was begun in March, 1939, and shortly thereafter a left intrapleural pneumonolysis was done. Left pleural effusion which was treated with aspiration followed; a few months later it became an empyema. Oleothorax was substituted for the pneumothorax in November, 1941, and the patient was dismissed in 1942.



A. *B.*

Fig. 1.—Case 5. *A*, Roentgenogram made six months prior to development of bronchopleural fistula. *B*, Roentgenogram made three days subsequent to development of bronchopleural fistula.

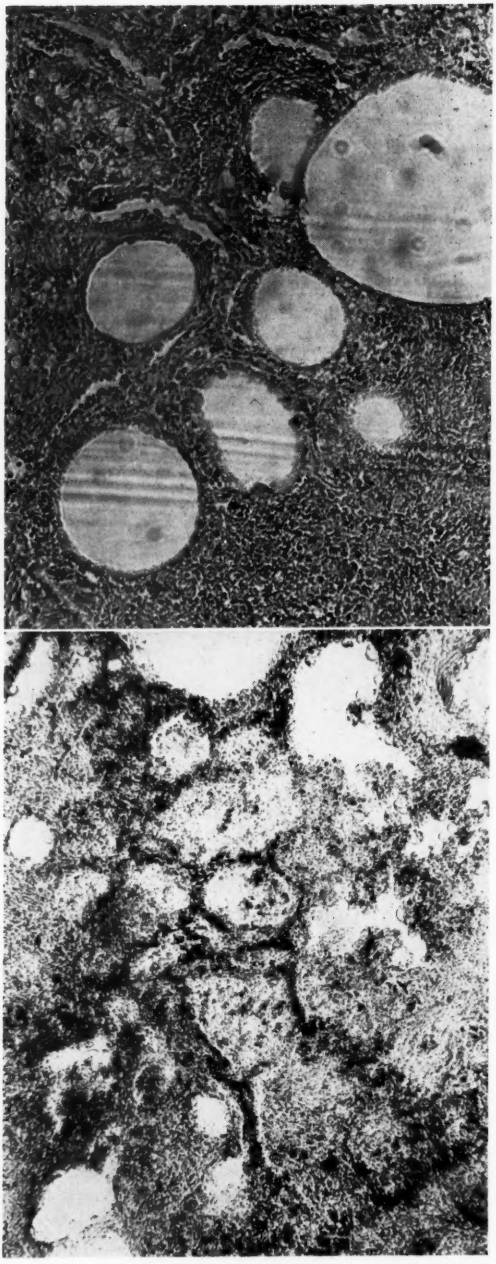
She did well until January, 1950, when she again began to cough and to have fever. Later she began to cough up oil. Readmission films revealed an extensive pneumonia-like process in the right lung (Fig. 1). The remaining oil was aspirated from the left chest, but the patient became markedly dyspneic and cyanotic and died March 18, 1950. Permission for autopsy was not obtained.

CASE 6.—W. M., a 29-year-old white laborer, was admitted to Nopeming Sanatorium in December, 1934, with far-advanced, bilateral pulmonary tuberculosis.

A left-sided pneumothorax was begun in January, 1934, and a right-sided pneumothorax was attempted but abandoned in October, 1937. The patient did well, however, and was discharged in June, 1940.

In December, 1943, he was readmitted with a left-sided reactivation. A decision to continue the pneumothorax was made, but tuberculous empyema soon ensued. The left pneumothorax was converted to oleothorax in September, 1945.

A.



B.

Fig. 2.—Case 6. *A*, Oil pneumonitis showing spaces filled with oil and granulomatous reaction in the surrounding tissue. *B*, Oil pneumonitis showing an extensive exudate and numerous globules of oil filling the alveoli. These stained bright orange with Sudan IV fat stain.

While on a leave of absence from the sanatorium in August, 1947, the patient developed a sudden coughing spell and coughed up profuse amounts of oil and became quite dyspneic and cyanotic. He was readmitted to the sanatorium as an emergency but died a few days later.

Autopsy revealed chronic, bilateral pulmonary tuberculosis with multiple cavities. A contributory cause of death was lipoid pneumonia throughout both lungs (Fig. 2). The diagnosis was proved by appropriate fat-staining methods.

CASE 7.—M. G., a 25-year-old single woman, was first admitted to Nopeming Sanatorium Aug. 5, 1934, with moderately advanced, bilateral pulmonary tuberculosis. There was multiple cavitation on the right with a fibrocaseous-appearing lesion on the left. Several attempts to begin a pneumothorax were unsuccessful.

On April 22, 1935, a right phrenic crush was done. Improvement was noted throughout the next two years, but in 1937, reactivation occurred. Because of this a left pneumothorax was begun. A left intrapleural pneumonolysis was done on Jan. 17, 1938; a right extrapleural pneumonolysis was done in March, 1938, and a paraffin pack was inserted into the right extrapleural space the following June.

The patient convalesced well and returned to work, but in August, 1941, she suddenly experienced pain in the right upper chest and neck, and shortly thereafter a mass in the upper anterior chest wall was detected. This increased in size for a day and then became stationary.

On examination a hard mass 3 to 4 cm. in its greatest dimension was palpable in the second anterior interspace in the right midclavicular line. At this time it was decided that surgery should not be done, but several months later when the mass had increased in size and there was more marked tenderness, in addition to its being obvious that the muscles and breast were being infiltrated, it was felt that removal of the pack was indicated.

On Feb. 14, 1942, the anticipated surgery was begun, but only the extruded portion of paraffin was removed. However, recurrence of the mass and symptoms necessitated reoperation in September, 1942, with removal of the entire pack at that time.

The patient was last seen in 1946, but prior to that time she had progressed well.

DISCUSSION

Although there is no doubt that plombage of the lung with pulmonary tuberculosis is of occasional value, in recent years methods have been developed which relegate such therapy to use in only a few well-selected cases. It seems, therefore, that the use of mineral oil or paraffin in the chest should be abandoned except for the rare case—as indeed it has been by most, primarily because there are more effective ways of treating the majority of cases of pulmonary tuberculosis. In the second place, the ever present danger of complications due to the foreign body reaction, which the agent itself will often produce, is a further argument in favor of the abandonment of the use of mineral oil and paraffin.

In our seven cases complications took the form of pleurocutaneous fistula, chest wall granuloma formation, and oil pneumonitis due to aspiration following bronchopleural fistula formation, the incidence of such complications being approximately 10 per cent.

There seems to be no evidence that chemotherapy or better technique would have altered or will in the future alter the situation regarding the use of mineral oil in the treatment of tuberculosis except to outmode it further.

CONCLUSIONS

1. Mineral oil and paraffin, when introduced into the human tissues, act as foreign agents. This is true in the case of oleothorax, but the exact incidence of granuloma formation in such cases is unknown. In our experience the incidence is about 10 per cent.

2. Chemotherapy and newer surgical techniques make the use of mineral oil and paraffin in the treatment of tuberculosis a rare necessity.

3. Not only does the development of granuloma along with migration restrict the use of mineral oil, but as a form of treatment per se, in comparison with other methods of treatment of pulmonary tuberculosis, it is not of great value.

REFERENCES

1. Adams, W. E.: The Use of Paraffin as an Aid in Obliterating the Pleural Space Following Total Pneumonectomy, Ann. Surg. **136:** 18-26, 1952.
2. Cutler, J. W.: Importance of Extrapleural Pneumothorax in Collapse Therapy of Pulmonary Tuberculosis, J. THORACIC SURG. **21:** 217-258, 1951.
3. Gaensler, E. A., and Strieder, J. W.: Progressive Changes in Pulmonary Function After Pneumonectomy: The Influence of Thoracoplasty, Pneumothorax, Oleothorax and Plastic Sponge Plombage on the Side of the Pneumonectomy, J. THORACIC SURG. **22:** 1-34, 1951.
4. Matson, R. W.: Oleothorax, Am. Rev. Tuberc. **25:** 419-468, 1932.
5. Browning, R. H., Ray, E. S., and Rotenberg, L.: Oleothorax: Re-evaluation With a Final Report of 101 Cases, Am. Rev. Tuberc. **54:** 122-127, 1946.
6. Ballantyne, A. J., Clagett, O. T., and McDonald, J. R.: Extrapulmonary Oil Granulomas Secondary to Oleothorax, Proc. Staff Meet., Mayo Clin. **27:** 250-256, 1952.
7. Kergin, F. G.: Esophageal Obstruction Due to Paraffinoma of the Mediastinum, Ann. Surg. **137:** 91-97, 1953.
8. Morutz, W., and Busanny-Caspari, W.: Quoted by Kergin.
9. Toussaint, P., and Toussaint, J. P.: Oleothorax and Paraffinomas, J. franç. méd. et chir. thorac. **3:** 301-307, 1949.
10. Krauss, N.: Freeing of the Esophagus Imbedded in Adhesions Due to Filling of the Pleural Cavity by Paraffin, Deutsche med. Wchnschr. **77:** 1144, 1952. Abstracted in Internat. Abstr. Surg. **97:** 137, 1953.
11. Nagel, O.: Contralateral Oil Pleurisy and Pericarditis in a Patient With Primary Oleothorax, Beitr. Klin. Tuberk. **103:** 82-89, 1950.

EXPERIENCES WITH THE CYTOLOGIC EXAMINATION OF BRONCHIAL SWABBINGS IN THE DIAGNOSIS OF CANCER OF THE LUNG

A Study of 602 Cases

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ACCURATE diagnosis of pulmonary cancer at the present time requires the combined efforts of the internist, the radiologist, the surgeon, and the pathologist. The greatest responsibility will always rest with the clinician who first studies the patient; suspicion of lung cancer and initiation of proper study are mandatory before any diagnostic team can function. Originally, the pathologist was limited to microscopic examination of small bits of tissue removed by bronchoscopic biopsy or expectorated by the patient. The pathologist's role in the diagnosis of carcinoma now is enhanced by the application of cytologic techniques; the significance of this method of tissue study is attested to by its widespread acceptance. The identification of cancer cells is a valuable aid to prompt diagnosis.

We have been interested in an uncomplicated technique that is well adapted to the cytologic study of fluids in general and that can be applied easily to the problem of diagnosis of carcinoma of the lung.

A method has been devised by one of us (J. B. H.) to study cells obtained by direct swabbing of the bronchial orifices at the time of diagnostic bronchoscopy. Examination of these simple wet-stained suspensions approximates the results of more complex procedures. The conventional methods that employ rapidly fixed and stained films from sputa or bronchial washings can be exacting and time consuming. We have found that prompt examination of the wet-stained suspensions obtained by direct bronchial swabbing is an accurate and economical method of cell study. Recently Jennings and Shaw[2] reported such a wet-film method used in studying sputum; the interested reader can find also a summary of most of the studies of bronchial cytology to date.

It is the purpose of this presentation to describe the details of the method and to relate the experiences with its use in the study of material obtained from 602 patients, each of whom was subjected to a single diagnostic bronchoscopy.

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MATERIAL AND METHODS

Technique.—

Preparation of staining fluid: The staining fluid used is similar to that previously employed for study of spinal fluid sediments[3] and is made in the following manner:

(National Aniline)

1. Toluidine blue (histologic) 1 Gm.
   Ethyl alcohol, 95% 20 c.c.
   Distilled water 80 c.c.
   Glacial acetic acid 1 c.c.
   Mortar the dry stain with the alcohol and slowly add the water. Transfer the resulting solution to a glass, stoppered bottle and add the acid.
2. Mix 10 c.c. of the toluidine blue solution (*1*) with 10 c.c. of human blood serum.
3. Centrifuge to remove the sediment. The supernatant fluid is decanted and comprises the staining fluid. It is kept in the refrigerator when not in use.

Procedure at bronchoscopy: A technician, specially trained in cytologic techniques, attends every diagnostic bronchoscopy. A single tray has been devised (D. B.) which is brought to the endoscopic room and contains the basic material necessary for preparation of specimens (Fig. 1).

Fig. 1.—Simple tray containing glass slides, forceps, stain, marking pencil, cover glasses, and acetic acid-alcohol fixative—all that is necessary for the technical part of the procedure.

Material is obtained through the bronchoscope by aspiration, by direct swabbing, or by biopsy. Occasionally washings are made and the aspirated fluid is transferred to vessels and sealed for centrifugation and study of the sediment. Small pledgets* attached to long applicators are used for swabbing

*Jackson sponge (Pilling Co.).

the suspicious area within the trachea or bronchus. These pledgets, first moistened in saline, are rubbed over the area selected and are then passed directly to the technician for immediate preparation.

Separate specimens are smeared and air dried for bacteriologic study.

Preparation of suspension of bronchial swabbings:

1. Place a large drop of staining fluid on a clean glass slide.
2. Remove the sponge from the bronchial sponge stick by grasping it tightly a few millimeters from the free margin with small forceps or snap. (This serves to isolate the surface covered with bronchial secretion from the unimportant yet absorbent portion of the sponge.)
3. Prepare the suspension by triturating the free margin of the sponge in a drop of staining fluid on a glass slide.
4. Apply a cover glass.
5. It is customary to prepare the material remaining on the sponge for examination by smearing it on a glass slide, fixing it immediately in acetic acid alcohol (95 per cent ethyl alcohol containing 3 per cent glacial acetic acid) and subsequently staining it by Papanicolaou's method.[4] This fixing agent has proved stable after repeated usage and does not evaporate as does the commonly used ether-alcohol solution. It is filtered before re-use. The rapidly fixed films are prepared last; the small amount of plasma absorbed by the sponge serves as an adhesive for remaining cells.

Usually two wet smears and two of the rapidly fixed type are prepared as described. The slides are then screened in the usual manner, utilizing the fresh preparations for immediate diagnosis. If no tumor cells are noted in the wet preparation, the fixed slides also are screened. In no case has the stained fixed film been positive and wet film negative.

The method, at first glance, would seem to lack provision of material for later review. However, it is a simple matter to separate the coverslip from the slide and drop both into acetic acid-alcohol fixative. The resulting fixed films subsequently can be stained as desired. The plasma-suspending medium serves as an excellent adhesive and the cells can be readily found after the slide and coverslip have been again mounted.

Histologic findings: The cytologic components of bronchial swabbings are similar to those seen in aspirations of bronchi. All types of bronchial epithelial cells are seen and readily identified (Fig. 2). Macrophages of varying stages of development are usually present; the histiocytes are sometimes difficult to interpret, as is the case with other cytologic techniques. The other types of inflammatory cells are seen in varying numbers. Lymphocytes in groups are of particular importance as they occasionally can be confused with the cells of small-celled carcinoma. The chromatin patterns and an irregularity of nuclear rims in the latter are valuable aids in differentiation.

The cytologic details that identify neoplastic elements in the wet suspension are the same as those that characterize these cells in the rapidly fixed films (Fig. 3). In the majority of cases tumor cells occur in clumps, a helpful feature for comparing aberrations in cell and nuclear size. Cellular hypertrophy alone is not considered diagnostic unless coupled with other findings. These changes include increased nuclear cytoplasmic ratio, anisocytosis, angulation of the nuclei and prominent nucleoli. Hyperchromatism is not a

prominent feature of neoplastic cells studied by this wet method, although irregular chromatin condensation is often apparent. Mitotic figures occasionally occur, but never are a prominent feature. A small-celled neoplasm can be difficult to diagnose, as only a few of the previously mentioned features may occur: namely, a tendency to exfoliate cells in groups, chromatin condensation, and nuclear angulation. These features serve to differentiate them from groups of lymphocytes and basal cells.

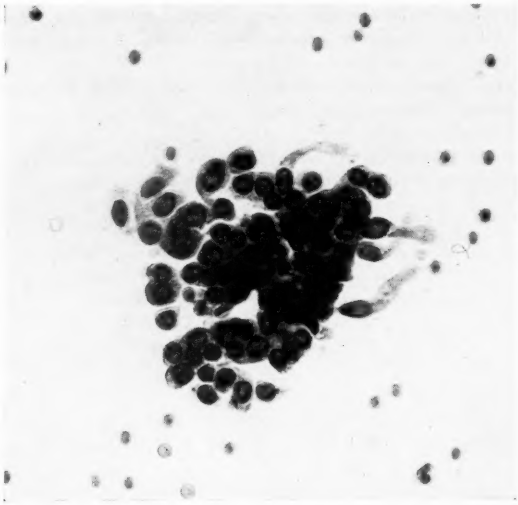

Fig. 2.—Clump of normal bronchial epithelial cells possessing all stages of maturation. (Serum-toluidine-blue stain; ×400.)

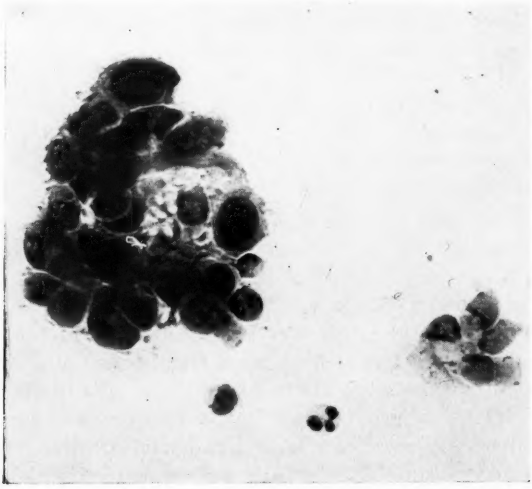

Fig. 3.—Neoplastic cells shed by a carcinoma of the bronchus. Note the hypertrophy of all elements, nuclear irregularity, and prominent nucleoli. A suggestion of pearl formation is present. (Serum-toluidine-blue stain; ×400.)

The method does not always permit identification of the cytologic types of the individual neoplasms which exfoliate large cells. Occasionally, faint evidences of "pearl" formations or a waxen eosinophilia of the tumor cells suggest the diagnosis of squamous-cell carcinoma but the red-to-orange colors of the Papanicolaou technique are not apparent. Vacuolated cells are seen in some cases of adenocarcinoma.

A general warning concerning this method of study is necessary. To us, the changes associated with malignancy are intensified in this type of preparation as compared with rapidly fixed wet films. All cells, including normal epithelial flora and histiocytes, are larger than the equivalent cells in fixed smears, e.g., young macrophages are larger than those seen in fixed smears. Consequently, the transition from the study of smears stained by the Papanicolaou technique to this wet method must be made carefully to avoid an erroneous diagnosis of carcinoma.

RESULTS

Between early 1950 and Sept. 1, 1953, the bronchial swabbings from 602 patients were examined at the Cleveland Clinic. Of this group, 200 had clinically definite carcinoma of the lung. Diagnoses were histologically substantiated in 79 per cent (158 cases) of the 200 cases, either by bronchial biopsy, surgical exploration, or lymph node biopsy. The remainder (42 cases) were accepted clinically as hopeless cases of carcinoma of the lung. The cytologic findings were positive for neoplasm in 47 per cent of the 200 cases, and there were no false positive reports during this period. However, we do not believe that this figure represents the highest accuracy of the method. In comparison with this percentage of the entire series, there was a 58 per cent evidence of positive results in the presence of neoplasm in the 400 most recent cases (1952 and 1953).

In contrast to the cytologic results, bronchial biopsy was positive in only 26 per cent of the total group and was constant throughout the series. Table I gives a comparison and summary of the results that can be obtained by utilizing the cytologic technique described as an adjunct to bronchoscopy, the latter essentially consisting of a single examination in each case.

TABLE I. COMPARISON OF BRONCHOSCOPIC BIOPSY AND CYTOLOGIC DIAGNOSIS OF CARCINOMA OF THE LUNG (200 CASES)

	NUMBER OF CASES	PER CENT
Cytology positive	93	47
Cytology only positive	55	28
Bronchial biopsy positive	52	26
Bronchial biopsy only positive	14	7
Both positive	38	19

In addition to the cases diagnosed as positive, the indeterminate diagnosis of the presence of suspicious cells was made in 23 of the 602 cases. Such a diagnosis is of some importance because 18 of the 23 cases were subsequently diagnosed as bronchogenic carcinoma. The remaining 5 patients suffered from some form of nonneoplastic chronic pulmonary disease.

COMMENT

It is apparent that information of definite value can be obtained by a simple method employed at the time of a single bronchoscopy. In this series no false positive diagnoses were made. The absolute reliability of a positive diagnosis is essential to the surgeon in those cases with the combination of a positive cytology, an indeterminate roentgenogram and clinically negative bronchoscopic findings.

Experience has led to an increased accuracy of the method, and the results now approach those reported for the more difficult method of examining multiple sputa.[5] The method, however, still lacks the sensitivity reported by Clerf and Herbut[1] in studying bronchial aspiration. In order to improve the sensitivity further, we believe multiple bronchoscopies would be necessary since the maximum effectiveness with a single specimen has been reached. Unfortunately, absolute accuracy is still not possible with any bronchoscopic and cytologic method; the responsibility for making the final evaluation of the case with negative cytologic findings rests with the clinician in charge of a patient.

SUMMARY

A simplified method of cytologic examination employing wet, fresh films prepared and studied as such from bronchial swabbings is described. The method is of no additional inconvenience to the patient and permits the pathologist to fix the preparation permanently for later or review examination.

Six hundred and two patients were so studied.

Of these, 200 were regarded clinically as having primary carcinoma of the lung; among these 158 (79 per cent) were histologically proved. The cytologic examination was positive in 93 (47 per cent) of this series of 200. An additional one-third of the cases can be diagnosed positively prior to surgical intervention as compared with the results of bronchoscopic biopsy alone.

With a combination of bronchoscopic biopsy and cytologic study in the manner described, two-thirds of the cases can be diagnosed positively, as evidenced by our results in studying the last 400 patients.

No false positive diagnoses were encountered in this series.

REFERENCES

1. Clerf, L. H., and Herbut, P. A.: Value of Cytological Diagnosis of Pulmonary Malignancy, Am. Rev. Tubere. **61**: 60-65, 1950.
2. Jennings, R. C., and Shaw, K. M.: Value of Cytologic Examination of the Sputum in the Diagnosis of Carcinoma of the Bronchus, Thorax **8**: 288-294, 1953.
3. McCormack, L. J., Hazard, J. B., Gardner, W. J., and Klotz, J. G.: Cerebrospinal Fluid Changes in Secondary Carcinoma of Meninges, Am. J. Clin. Path. **23**: 470-478, 1953.
4. Papanicolaou, G. N.: Atlas of Exfoliative Cytology, Cambridge, Mass., 1954, Harvard University Press, p. 6.
5. Woolner, L. B., and McDonald, J. R.: Cytologic Diagnosis of Bronchogenic Carcinoma, Am. J. Clin. Path. **19**: 765-769, 1949.

THE MIDDLE LOBE SYNDROME AND ITS RELATIONSHIP TO CERTAIN ASPECTS OF MIDDLE LOBE DISEASE

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IN 1948 the term *middle lobe syndrome* was introduced by Graham, Burford, and Mayer,[1] to categorize a condition resulting from compression of the middle lobe bronchus by enlarged peribronchial lymph nodes with secondary changes in the distal parenchyma. It is now recognized clinically to be of rather frequent occurrence, and the term has gained wide usage. The exact definition of the middle lobe syndrome and the interpretation of its relationship to certain conditions affecting the middle lobe appear to be somewhat ambiguous. In order to ensure proper management, a full understanding of this condition and certain primary diseases of the middle lobe simulating it is essential.

The particular vulnerability of the right middle lobe bronchus was first described by Brock[2-4] on the anatomic basis of its length and narrowness, its acute angle of origin from the bronchus intermedius, and its close relationship to lymph nodes that drain the right lower lobe as well as the middle lobe and other lobes. He noted the frequency with which the enlarged hilar nodes of the primary tuberculous complex compressed and eroded the relatively soft middle lobe bronchus in infants and children. This concept of etiological association with tuberculosis soon became established and persists today. In 1946 Zdansky[5] called attention to a nontuberculous etiology of the condition by presenting evidence of nonspecific lymphadenitis obstructing the middle lobe bronchus. Two years later, Graham, Burford, and Mayer[1] described twelve cases of middle lobe bronchial compression by lymphadenopathy not caused by tuberculosis. References to the syndrome[6-10] appeared with increased frequency following that report.

In our recent experiences with the condition treated surgically at Fitzsimons Army Hospital, we have encountered three cases caused by histoplasmosis and one by sarcoidosis. Such unusual etiology stimulated a perusal of literature pertaining to the middle lobe syndrome. After reviewing all cases in this hospital treated by middle lobectomy from 1946 to 1953, we were struck by the difficulty of defining an absolute classification of the syndrome so as to encompass the many variables encountered in each individual. Indeed, the complete picture can often be understood only when all of the varying factors are brought into proper perspective by the clinician, surgeon, and pathologist. The presenting symptomatology may be alike in most cases, conveniently allowing classification as a separate syndrome, whereas, in truth, the underly-

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ing pathologic agents affecting the middle lobe may be of considerable diversity. In fact, in the final analysis of the entire picture it may become apparent that a case originally diagnosed as middle lobe syndrome does not completely satisfy the classical criteria of bronchostenosis with perihilar lymphadenopathy demanded by strict definition and is, in reality, a type of middle lobe disease that closely resembles the syndrome. Five cases selected from this series are presented to illustrate certain features encountered in patients with the middle lobe syndrome and certain related diseases of the middle lobe.

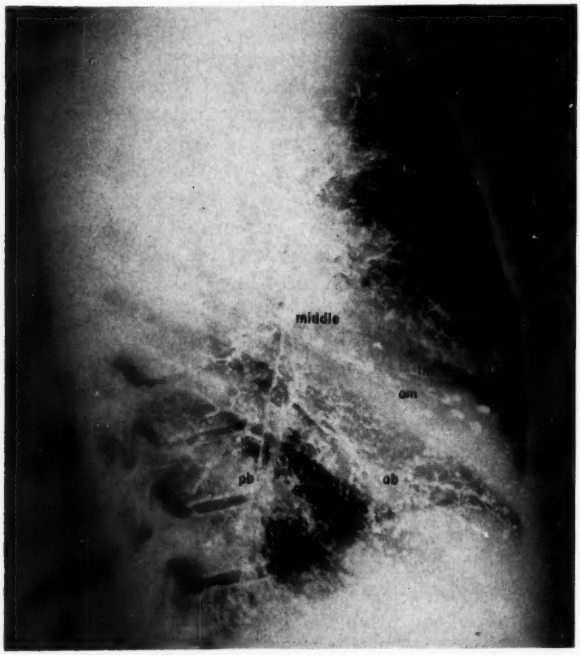


Fig. 1.—Case 1. Lateral bronchogram showing contracted middle lobe with cylindrical and saccular bronchiectasis, but no bronchostenosis.

CASE REPORTS

CASE 1.—A 19-year-old white man was admitted to this hospital with a history of chronic productive cough, frequent colds, and occasional right lower anterolateral pleuritic pain as long as he could remember. He had had severe right-sided pneumonia when 5 years old and again when 13 years old. A chest x-ray shortly preceding this admission showed right middle lobe pneumonitis. On admission he was raising one-half cup of greenish-yellow mucopurulent sputum daily. Similar material was seen coming from the right middle lobe orifice bronchoscopically. Numerous smears and cultures were negative for Mycobacteria. A bronchogram demonstrated saccular bronchiectasis with crowding of the distal middle lobe bronchi (Fig. 1). All other bronchi appeared normal. On Jan. 18, 1949, thoracotomy revealed normal right upper and right lower lobes, but there were very dense adhesions and scarring about the right middle lobe hilus with bronchial thickening. No peribronchial lymphadenopathy was noted. Recovery, following right middle lobectomy, was uneventful.

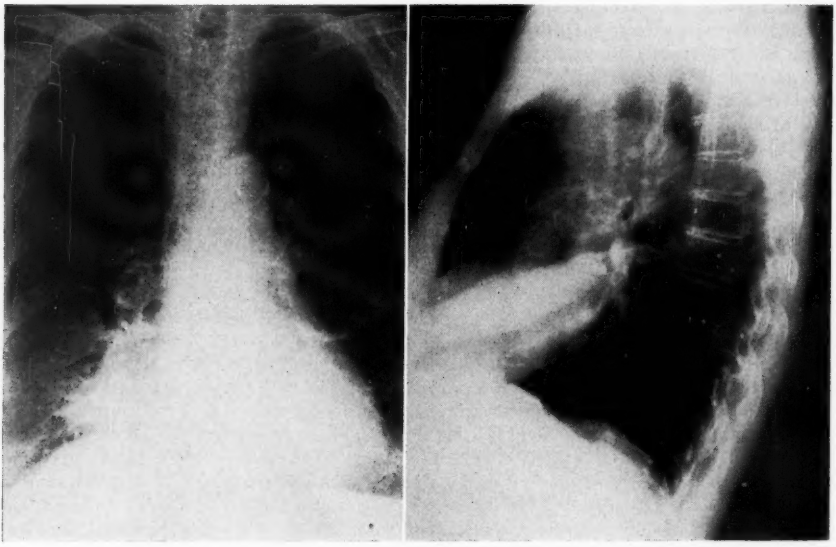
Pathologic Examination.—The lobe was moderately reduced in volume. No parenchymal defects were apparent grossly, although thickened dilated bronchi were palpable. The middle lobe bronchus had been transected at its carina, and gross mural thickening was evident at the origin of the medial and lateral segmental bronchi. No lymph nodes were seen, but microscopic sections disclosed residuals of lymphoid tissues closely applied to the major bronchial walls at their resection margins which, presumably, were contiguous with lymphoid tissue adherent to the proximal stump. These showed evidence of scarring and reactive hyperplasia consistent with chronic nonspecific inflammation. There was marked tubular and saccular bronchiectasis of classical variety, particularly of the lateral segment. The lumina measured as much as 2.0 cm. in circumference. Abundant evidence of active acute and chronic nonspecific bronchitis was obtained; one focus of mucosal ulceration was found deep within a branch of the middle segmental bronchus. Numerous foci of chronic bronchiolitis, interstitial pneumonia, and scarring were scattered throughout the pulmonary parenchyma. Cultures were negative for Mycobacteria and pyogenic organisms.

Comment.—This case represents an example of bronchiectasis of the middle lobe that is frequently referred to as the middle lobe syndrome. Certainly most of the clinical features are suggestive of the syndrome, but without bronchostenosis and peribronchial lymphadenopathy such a diagnosis is untenable in the strict sense. It could be postulated that the changes exhibited are only the result of insults from the childhood pneumonia. However, it also could be argued that the past pneumonic episodes resulted in acute peribronchial lymphadenitis. This, perhaps with the edema of local bronchitis, produced temporary bronchostenosis and was predisposed to stasis, secondary bronchitis, and bronchiolitis within the lobe. Chronic interstitial pneumonitis became established, which, with scarring, contributed to the perpetuation of the bronchiectasis. The latter predisposed to recurrent bronchial infection long after the local hilar bronchial disease had subsided; thus a vicious cycle was initiated. The peribronchial lymphoid tissue seen on microscopic section could represent the residuals of the previously involved lymph nodes. Despite the attractiveness of this hypothetical pathogenesis, the available information necessitates a diagnosis of bronchiectasis of the middle lobe rather than a true middle lobe syndrome.

CASE 2.—A 75-year-old white woman entered the hospital with a four- to six-month history of a hacking cough productive of one teaspoon of yellow sputum daily, heavy aching over lower right anterior chest, and a 10-pound weight loss associated with anorexia and increased fatigability. The past history was not contributory. A poorly circumscribed right hilar mass could be seen on x-ray in association with an atelectatic right middle lobe (Fig. 2). Repeated bronchoscopies revealed an indefinite mass in the right middle lobe bronchus. Biopsies were reported as chronic bronchitis and squamous cell metaplasia, but never definitely as neoplasm. Atypical cells were noted on Papanicolaou smears. Repeated cultures of sputum and bronchial washings were negative for tuberculosis and fungi. A tuberculin skin test was negative. Extensive antibiotic treatment had no effect. Exploratory thoracotomy was performed Dec. 8, 1952, with a preoperative diagnosis of probable carcinoma of the middle lobe. The middle lobe was found to be contracted and almost leathery in consistency. Several enlarged calcified lymph nodes were present in the right hilum and especially marked around the middle lobe bronchus. No neoplasm could be demonstrated, and a middle lobectomy was performed. For technical reasons the main middle lobe bronchus was divided distal to the densely adherent collar of enlarged peribronchial nodes. The patient was discharged improved after an essentially uneventful postoperative course.

Pathologic Examination.—The lobe was markedly shrunken, noncrepitant, and fibrous. A few fibrous adhesions were present, but the pleura was not remarkable otherwise. Resection had been accomplished a few millimeters proximal to the principal carina of the middle lobe, and no lymph nodes were adherent to the stump from which the nature of the lymphadenopathy described at surgery could be determined. The bronchi were uni-

formly dilated in tubular fashion and filled with thick, mucopurulent exudate. Extreme chronic nonspecific inflammatory reaction involved the bronchial mucosa, and there was marked transverse fibrous ridging. The parenchyma was diffusely atelectatic and had undergone considerable interstitial fibrosis. There was extensive bronchiolitis with active organizing interstitial pneumonia in the surrounding parenchyma. Cultures for pyogenic, acid-fast, and mycotic organisms remained sterile.



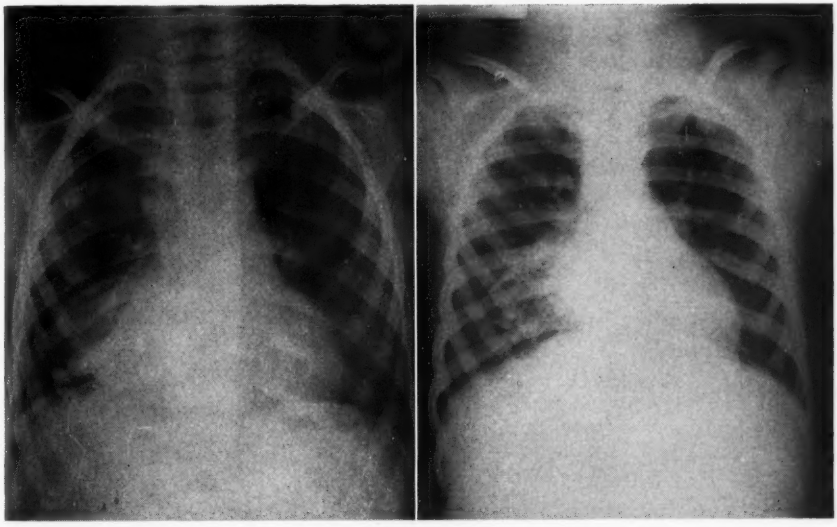
A. *B.*

Fig. 2.—Case 2. *A* and *B*, Roentgenograms of atelectatic middle lobe. Note classical picture on lateral projection.

Comment.—This case emphasizes the necessity for providing the pathologist with adequate material if etiological diagnosis is to be obtained. The surgeon elected to divide the middle lobe bronchus distal to the density adherent collar of nodes and did not remove a node for examination. The finding of calcifications in the middle lobe hilar lymph nodes is strong presumptive evidence that a granulomatous disease existed, but whether this represents tuberculosis, histoplasmosis, coccidiomycosis or a more esoteric infection probably will never be known. The case, however, illustrates the extent to which the middle lobe is susceptible to extrinsic lymph node disease and the degree of secretory retention which may result from incomplete obstruction. The absence of sacculation suggests that the obstructive process was not of protracted duration and supports the possibility that active residual inflammatory disease persisted in the hilar lymph nodes. The extrinsic atelectasis must be ascribed to the retained secretions rather than directly to the partially stenosed stem bronchus. The short history in a patient of this age group in association with the other findings strongly suggests bronchogenic neoplasm. In all such cases, the question of malignancy finally must be settled by surgical exploration.

CASE 3.—A 4-year-old white girl was admitted to the hospital in November, 1951. A twin brother had died of tuberculous meningitis in May, 1951. The patient's chest x-ray at that time was interpreted as showing right middle lobe atelectasis, probably nontuberculous. Since gastric washings were negative, the child remained at home on bed rest. The appearance of the chest was unchanged by x-ray over the next five months. A repeat gastric washing was reported tuberculosis positive on guinea pig inoculation in October,

1951. Streptomycin, 0.5 Gm. every three days, and para-aminosalicylic acid (PAS), 5 Gm. daily, were started. The child was alert and active, but wheezing and occasional cough were noted. The chest was flattened anteriorly on the right with diminished breath sounds. Admission chest x-rays showed hilar adenopathy, most marked on the right, with atelectasis of the right middle lobe (Fig. 3, *A*). Repeated bronchoscopies were performed between November, 1951, and June, 1953. Marked distortion of the right bronchial tree with narrowing was noted. Initially, penetrating caseous lymph nodes were removed bronchoscopically to enlarge the lumen of the right main bronchus. The endobronchial changes slowly improved, but some irreversible damage persisted. Subsequently, a bronchogram revealed good filling of all segments with bronchiectasis of the right middle lobe and anterior segment of the right upper lobe. The hilar adenopathy had subsided by x-ray prior to surgery (Fig. 3, *B*). On June 19, 1953, the right middle lobe and anterior segment of the right upper lobe were resected. Both were atelectatic and fibrotic. Dissection of the atelectatic fibrotic diseased parenchyma was difficult because of the extensive peribronchial scarring. However, a careful search failed to reveal definite hilar adenopathy. An 8 mm. primary complex was identified in the right lower lobe. The postoperative course was uneventful. Streptomycin and PAS were continued throughout the hospitalization for a total of nineteen months.

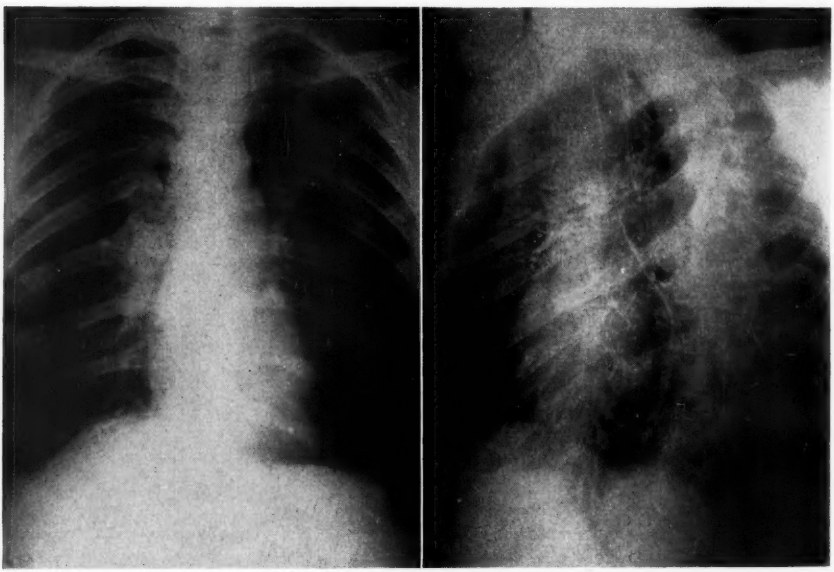


A. *B.*

Fig. 3.—Case 3. *A*, Roentgenogram on admission. Note hilar adenopathy and opacified middle lobe. *B*, Roentgenogram shortly before surgery. The hilar adenopathy has regressed after prolonged streptomycin and para-aminosalicylic treatment.

Pathologic Examination.—The middle lobe was markedly shrunken and had no pleural lesions. The bronchi displayed marked tubular and saccular bronchiectasis with moderate trabeculation. That of the medial segment was significantly distended with mucus. All exhibited variable mucosal atrophy, scarring, and intense nonspecific inflammatory reaction. In addition to extensive chronic nonspecific bronchiolitis, the smaller air passages frequently were distended with retained mucus. Gross and microscopic examination of the parenchyma disclosed well-developed atelectasis and focal areas of chronic interstitial pneumonitis undergoing organization. No tuberculoid lesions were encountered throughout, and bacteriologic studies were negative for *Myco. tuberculosis*, fungi, and other pathogens. The only diagnosis permissible on pathologic examination was that of severe chronic nonspecific bronchiectasis.

Comment.—The hilar lymphadenopathy associated with primary tuberculosis not uncommonly causes bronchial distortion and compression. In infants and children, in particular, caseous lymph nodes may progressively erode the bronchial wall to such an extent that endoscopic removal is necessary to maintain an adequate airway. Such a situation was present in this child. It was of interest to note that after nineteen months of continuous sterptomycin and para-aminosalicylic acid, no evidence of the caseous hilar adenopathy was noted on gross inspection at thoracotomy. In fact, to the pathologist receiving the resected pulmonary tissue, there were no absolute grounds upon which to base a diagnosis of tuberculosis. The subpleural focus identified by the surgeon in the right lower lobe, presumably the parenchymal component of the primary complex, might have served this purpose had it been removed for histologic examination. In the same way, lymphoid residuals occasionally have been demonstrated in such matted perihalar substance. The clinical information available, nevertheless, suggests tuberculosis as the probable etiological agent. This case illustrates the nonspecific destructive changes that may remain when the original disease that initiated the hilar lymphadenopathy is no longer apparent. Less severe cases that would have escaped surgical attention at this early stage may present themselves in later years as the middle lobe syndrome or nonspecific middle lobe inflammatory disease. It is not uncommon to see residual destruction in other lobes, such as was demonstrated in the anterior segment of the right upper lobe in this case.



A. *B.*

Fig. 4.—Case 4. *A*, Roentgenogram with right hilar adenopathy due to sarcoidosis. *B*, Bronchogram showing stenosis of right middle lobe bronchus secondary to lymphadenopathy.

CASE 4.—A 26-year-old white soldier was admitted to this hospital with a six-year history of repeated colds and upper respiratory infections and a chronic cough productive at times of white and yellow sputum. He was hospitalized twice during the previous one and one-half years because of pneumonia on the right side. Recently, the cough had become more productive, and blood-tinged sputum had been noted. Bronchitis was thought to be the underlying cause of his respiratory complaints.

On admission, he did not appear acutely ill or dyspneic. No cervical nodes were noted, but the spleen could be palpated two fingerbreadths beneath the costal margin. Skin tests for tuberculosis, coccidioidomycosis, and histoplasmosis were negative; all other laboratory tests were noncontributory. Chest x-ray and fluoroscopy revealed increased prominence of the right hilum and a widened mediastinum (Fig. 4, *A*). The tracheobronchial tree appeared normal on bronchoscopy. Bronchograms showed stenosis of the right middle lobe bronchus; a repeat bronchogram confirmed this obstruction in the right middle lobe bronchus (Fig. 4, *B*). Palpation of the right lung at exploratory thoracotomy demonstrated fine diffuse infiltrations throughout, with many small nodules in the right middle lobe. Large boggy lymph nodes were present in the mediastinum. The extreme lymphadenopathy surrounding and compressing the right middle lobe bronchus made removal of this lobe technically difficult. The postoperative course following right middle lobectomy and mediastinal lymph node biopsy was uneventful.

Pathologic Examination.—The lobe was of nearly normal size. It appeared well aerated except for an area of atelectasis within the lateral segment adjacent to the hilus. Numerous smooth 1 mm. waxy nodules were palpable and visible within the anterior portion. The pleura appeared smooth and uninflamed. The lateral segmental bronchus and its immediate branches were stenosed by dense fibrous tissue throughout which were scattered numerous noncaseating naked tubercles consistent with sarcoid lesions. The remaining bronchi showed minimal tubular dilations and little or no inflammation other than scattered mucosal sacroid lesions. The gross parenchymal nodules were identical histologically, containing numerous granulomas in perivascular and peribronchial pattern. Dense bands of fibrous tissue interconnected the lesions. The intervening alveolar parenchyma disclosed patchy atelectasis and emphysema. The several hilar lymph nodes which were received separately were enlarged to 1.5 cm. but were relatively discrete and likewise contained numerous noncaseating tubercles. Asteroid and Schaumann bodies were encountered rarely within the Langhans giant cells which populated the granulomas in large numbers. Direct smears, appropriately stained sections, and cultures failed to disclose the presence of tubercle bacilli, fungi or pyogenic organisms. A diagnosis of pulmonary and lymph node sarcoidosis was made without reservation.

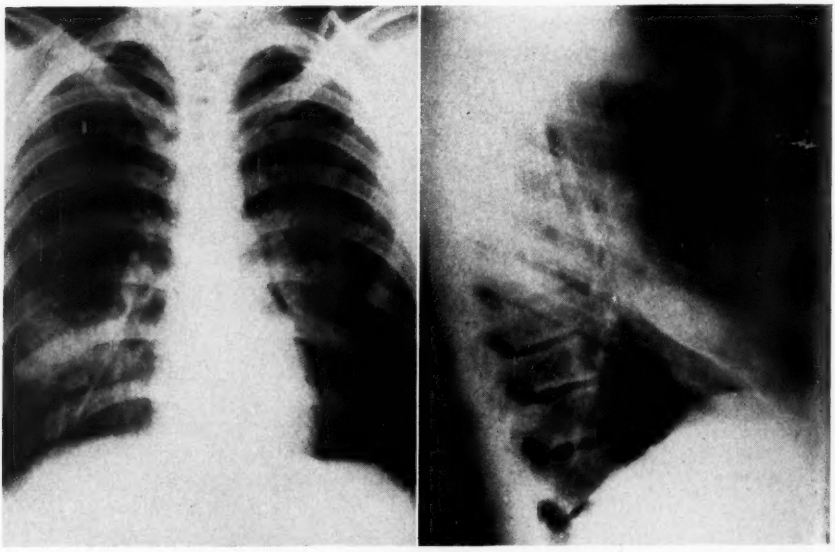
Comment.—This case is the first, to our knowledge, in which sarcoidosis has been etiologically significant in the production of the middle lobe syndrome. Focal intralobar atelectasis is mentioned briefly by Longcope and Freiman[11] in their monumental discussion of this disease, but no mention is made of compression of a major bronchus. This well may be due to the lack of perilymphadenitis and the relative absence of matting of lymph nodes considered characteristic. In this instance, the obstruction is considered mild and probably intermittent as reflected by the relatively insignificant parenchymal alterations. That it was clinically important, however, is reflected by the recurrent pneumonitis. With the exception of the well-developed scarring, the pulmonary changes are considered to be reversible.

CASE 5.—A 22-year-old white soldier was admitted to this hospital because of five episodes of right middle lobe pneumonia during the previous two years, that required hospitalization and extensive antibiotic treatment on each occasion. Each attack was characterized by malaise, chills, fever, and right pleurtic pain with x-ray evidence of right middle lobe pneumonitis. The past history otherwise was not remarkable. On admission here, he was afebrile and appeared to be healthy. Physical examination was essentially negative. Skin tests were positive for histoplasmosis and negative for tuberculosis and coccidioidomycosis. Repeated cultures of sputum and bronchoscopic washings were negative for acid-fast bacilli, fungi, and tumor cells. X-ray examination of the chest disclosed soft, fluffy infiltrations in the right middle lobe suggesting pneumonitis (Fig. 5). Bronchoscopy failed to reveal any abnormality, but by bronchogram the medial segment of the right middle lobe was noted to be irregular and bronchiectatic.

At thoracotomy, Feb. 26, 1954, the right upper and lower lobes were normal, but the middle lobe felt indurated. The major fissure was partially obliterated by adhesions.

A collar of densely adherent lymph nodes presented about the main middle lobe bronchus. To divide this bronchus, it was necessary to transect an adherent grossly granulomatous node wedged in the junction with the bronchus intermedius. The postoperative course following middle lobectomy was uneventful.

Pathologic Examination.—The middle lobe was moderately shrunken. The pleural surface was roughened by numerous fibrous adhesions presumably reflecting the numerous attacks of pneumonitis. The middle lobe bronchus had been severed 5 mm. proximal to its primary carina, and adherent to the medial aspect of the stump were three small lymph nodes matted together into a single mass. They contained numerous discrete yellow granulomas from which caseous material could be expressed. Histologic study revealed these lesions to be of tuberculoid variety, surrounded by dense fibrous tissue. Epithelioid cells, plasma cells, lymphocytes, and Langhans giant cells were noted at their periphery, and their centers were composed of amorphous caseous debris. Moderate fibrosis of the surrounding parenchyma had occurred. Smears, cultures, and tissue sections were negative for tubercle bacilli, but sections stained by the periodic acid-Schiff method contained small clusters of minute spheroidal organisms with a central punctum of chromatin and a thin capsule which were morphologically identical to *Histoplasma capsulatum* (Fig. 6). These appeared somewhat distorted, swollen, and sometimes calcified, suggesting nonviability. Cultures failed to yield fungi.



A. *B.*

Fig. 5.—Case 5. *A* and *B*, Roentgenograms showing nonspecific middle lobe pneumonitis secondary to histoplasmosis lymphadenopathy.

The lymph nodes, although not significantly adherent to it, produced visible compression of the medial segmental bronchus, the branches of which exhibited a moderate degree of tubular and saccular bronchiectasis and chronic nonspecific inflammation. Minimal chronic inflammatory and bronchiectatic changes were present in the lateral segment. The pulmonary parenchyma, particularly of the medial segment, was the site of mild patchy bronchiolitis and chronic nonspecific interstitial pneumonitis, a few of which were palpable. Some of these contained an alveolar exudate including cholesterol-type macrophages consistent with obstructive etiology.

Comment.—This case emphasizes the value of careful examination of obstructing lymph nodes in determining etiology and illustrates the necessity for more complete study of lesions bearing morphologic identity with tuberculosis, but in which acid-fast organisms cannot be found. In two similar instances, histoplasma lymphadenitis and middle lobe disease, pathogenetically related to the middle lobe syndrome, have been seen by us. In one, a calcified node had ulcerated into the main middle lobe bronchus and presented to the surgeon as a broncholith when the bronchus was transected. In the other, extension of the infection had occurred in the region of the middle lobe hilus resulting in a broad focus of granulomatous pneumonia. It is felt that *Histoplasma capsulatum* is not generally appreciated as a cause of hilar lymphadenopathy capable of bronchial obstruction.

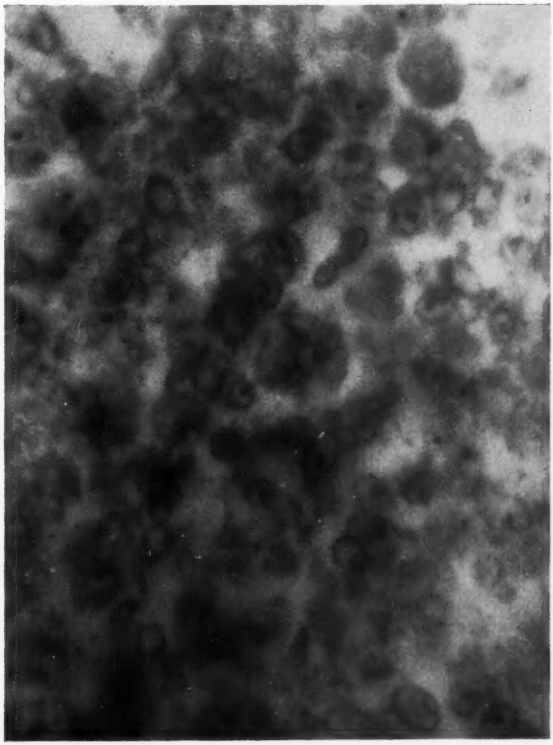


Fig. 6.—Case 5. *Histoplasma capsulatum* in lymph node.

DISCUSSION

Fundamentally, the middle lobe syndrome is the resultant of variable interplay between lymph nodes, bronchus, and distal lung parenchyma. The middle lobe bronchus is in the drainage pathway from the middle lobe and lower lobe parenchyma. The lymphadenopathy may be acute in nature and result in temporary mechanical bronchial compression which resolves more or less completely, leaving no permanent parenchymal damage. Epituberculosis, so-called, or an acute respiratory infection in a child may present such

a picture. If acute bronchitis occurs secondary to the lymphadenitis, inflammatory edema may further narrow the mechanically compressed middle lobe bronchus. If the infection is severe with peribronchial inflammation, permanent scarring and fibrosis of the peribronchial structures may result. This point is well demonstrated in Case 1. Severe peribronchial inflammation of this degree usually also involves the bronchial wall to such an extent that a poorly functioning or narrowed bronchus results, thus preparing the way for future infections. The acute caseous tuberculous nodes seen in childhood sometimes erode and penetrate the bronchial wall, resulting ultimately in changes in caliber in the bronchus as seen in Case 3. The more chronic calcified type of node seen in histoplasmosis as well as tuberculosis may also erode the wall, resulting in bleeding, further infection, or true broncholiths.

The nature of the pathologic changes in the distal bronchi and parenchyma depend upon such factors as the nature and duration of the obstruction and the presence or absence of secondary infection in the obstructed distal lung. If infection does not supervene, an atelectatic middle lobe may be an incidental finding on survey chest x-ray. As indicated above, the more common course is that of repeated bouts of infection leading to persistent lymphadenopathy, further bronchial compression, and increased obstruction resulting in a truly vicious pathologic and clinical cycle. Each repeated episode can only result in increasingly severe damage to parenchyma and bronchus, with ultimately the findings of gross bronchiectasis, chronic pneumonitis, fibrosis, and even lung abscess, bronchopleural fistula, and empyema.

The clinical diagnosis of middle lobe syndrome often means little in itself and can only lull the clinician into a false sense of security unless he is aware of the numerous etiological agents that may operate. The patient may present with cough, sputum, hemoptysis, and recurring pneumonitis, the classical symptoms of middle lobe syndrome, but the etiological agents responsible for the middle lobe abnormalities found clinically are variable. Although the pathogenetic mechanisms that produce the syndrome are fairly uniform, an understanding of the etiological agents is necessary in order to manage the case properly. In children, tuberculous hilar adenopathy is often responsible. MacPherson[12] lately has described a series of carefully studied cases of lung collapse associated with primary tuberculous lesions. Although carcinoma of the middle lobe is generally considered to be uncommon, Baldry[13] has recently reported a series of cases. The possibility of an underlying neoplasm simulating the complex must always be considered in the older age group, and exhaustive studies may still leave the diagnosis in doubt necessitating exploratory thoracotomy. This is demonstrated in Case 2.

Valle[14] has recently reported an unusual instance of middle lobe collapse due to hilar adenopathy related to inflammatory reaction secondary to lower esophageal trauma. Wohl, Israel, and Frobese[15] have described a patient with middle lobe syndrome that may have been due to histoplasmosis. The diagnosis was suggested by a positive skin test but unfortunately could not be confirmed by additional careful studies. Our three cases of histoplasmosis were established by careful search of tissue sections stained by the periodic acid-Schiff method after routine staining failed to demonstrate the

presence of organisms. Puckett[16] has emphasized the usefulness of this procedure in demonstrating the tissue phase of histoplasma. Exhaustive search for a fungus etiology when the surgeon provides adequate material undoubtedly allows specific diagnosis in many instances previously diagnosed as middle lobe syndrome due to granuloma of undetermined etiology.

The middle lobe syndrome comes to mind when the clinician is confronted with a story of repeated respiratory infections in the middle lobe area, productive cough, and hemoptysis. A careful history and thorough laboratory studies, including skin tests and cytology, may uncover the underlying etiology. The chest x-ray may vary with the underlying pathology from the characteristic appearance of a completely atelectatic middle lobe to that of acute pneumonitis. The right lateral projection may demonstrate changes easily missed on the anteroposterior film. The presence of hilar lymphadenopathy, especially with calcification, should be carefully noted. Bronchoscopy not only helps to evaluate stenosis and the origin of secretions or blood, but also permits a check for neoplasm, foreign body, perforating lymph nodes, and broncholiths. A bronchogram often shows obstruction, in the main or segmental middle lobe bronchus, beyond the vision of the bronchoscopist.

The thoracic surgeon sees numerous cases diagnosed middle lobe syndrome that require thoracotomy to establish a definite etiological agent. He is also fully cognizant of the many technical difficulties and potential hazards that may be associated with middle lobectomy in the presence of localized lymphadenopathy, perihilar interlobar fibrosis, and inflammatory reaction. The asymptomatic individual found to have a collapsed middle lobe by x-ray occasionally might be treated expectantly by a physican conversant with all aspects of the syndrome. Similarly the need for surgical relief of symptoms in the nonspecific cases may be tempered accordingly by such factors as age, operative risk, and the nature and severity of symptoms. In general, however, the treatment of this syndrome is essentially surgical; and only rarely, when a benign etiology is established and the lesion proved to be stable, can the physician afford to temporize by careful observation.

Because the term, middle lobe syndrome, is now well established and in wide usage, it becomes necessary to define accurately its meaning and to understand completely its relationship to certain diseases of the middle lobe that closely simulate it. Only in this way can existing ambiguity and confusion be dispelled and the proper management of patients so diagnosed be assured. Originally, the syndrome was defined to denote enlarged peribronchial lymph nodes compressing the middle lobe bronchus with certain changes in the distal parenchyma. Without these limitations, imposed by definition, the syndrome imperceptibly merges with a large number of other conditions affecting the middle lobe. Therefore, it is our contention that a diagnosis of middle lobe syndrome should not be made unless the following criteria have been met satisfactorily: (1) hilar or peribronchial lymphadenopathy, (2) bronchostenosis, and (3) the variable changes in the distal parenchyma, previously mentioned. It becomes apparent that the final interpretation may be possible only after reviewing the complete clinical, surgical, and pathologic findings.

Lymphadenopathy may have been demonstrated during the acute phase by x-ray or bronchoscopy and yet not be apparent at a somewhat later stage on gross or microscopic examination. In the same way, bronchoscopy and/or bronchography may have shown middle lobe bronchostenosis that proves to be reversible when the acute phase of pneumonitis, lymphadenitis, and bronchitis subsides. In these somewhat borderline examples of the syndrome, one can appreciate the need for a complete evaluation of *all* data without which the condition could be variably interpreted by internist, surgeon, and pathologist.

These alterations initiated by peribronchial lymphadenopathy may involve other lobes and segments independently or concurrently with the middle lobe. The middle lobe, of course, is most commonly involved; but when, in addition, several segments or lobes are affected, we believe the condition no longer fits the true definition of middle lobe syndrome.

As previously mentioned, the term middle lobe syndrome has become established by usage and will probably persist. Rather than belabor the need for this questionable separate designation, we feel that the problem can be met best by accurately defining the syndrome and correctly interpreting it and its relation to certain other middle lobe conditions. In this way the term will take on a more definite meaning and form a more concrete basis upon which to establish definitive treatment.

SUMMARY

The term middle lobe syndrome is defined, and its relationship to certain diseases of the middle lobe that may simulate it discussed. The wide use of the term demands a clarification of the existing ambiguity of definition and interpretation so as to ensure proper management of the individual so diagnosed. The syndrome must meet the following criteria in reference to the middle lobe: (1) hilar or peribronchial lymphadenopathy, (2) bronchostenosis, and (3) certain changes in the distal parenchyma discussed in the body of the paper. The final interpretation is often possible only after the complete clinical, surgical, and pathologic data are reviewed.

Special stress is laid upon the diversified etiology that may be present in contrast to a more constant symptomatology. The concept, so strongly held in the past, of commonly associating the syndrome with tuberculosis must be abandoned. Selected case reports of middle lobe syndrome and closely related middle lobe disease are included to form a basis for discussion of certain aspects of the problem. Among these are examples due to sarcoidosis, histoplasmosis, tuberculosis, and nonspecific etiology.

REFERENCES

1. Graham, E. A., Burford, T. H., and Mayer, J. A.: Middle Lobe Syndrome, Postgrad. Med. **4**: 29, 1948.
2. Brock, R. C., Cann, R. J., and Dickinson, J. R.: Tuberculous Mediastinal Lymphadenitis in Childhood. Secondary Effects on Lungs, Guy's Hosp. Rep. **87**: 295, 1937.
3. Brock, R. C.: Post-tuberculous Broncho-stenosis and Bronchiectasis of the Middle Lobe, Thorax **5**: 5, 1950.
4. Brock, R. C.: The Anatomy of the Bronchial Tree Wth Special Reference to the Surgery of Lung Abscess, London, 1946, Oxford Medical Publications.
5. Zdansky, E.: Der Mittlelappen Als Punctum Minoris Resistentia Der Lung, Wien. klin. Wchnschr. **58**: 197, 1946.

6. Paulson, D. L., and Shaw, R. R.: Chronic Atelectasis and Pneumonitis of the Middle Lobe, J. THORACIC SURG. **18**: 747, 1949.
7. Rubin, E. H., and Rubin, M.: The Shrunken Right Middle Lobe With Reference to the So-called "Middle Lobe Syndrome," Dis. Chest **18**: 127, 1950.
8. Bryant, J. R., and Harter, J. S.: Middle Lobe Disease, Dis. Chest **18**: 250, 1950.
9. Dross, I., and Rosenblatt, M.: The Middle Lobe Syndrome, J. Mt. Sinai Hosp. **17**: 711, 1951.
10. Frethein, B.: The So-called Middle Lobe Syndrome, Thorax **7**: 156, 1952.
11. Longcope, W. T., and Freiman, D. G.: A Study of Sarcoidosis, Medicine **31**: 1, 1952.
12. MacPherson, A. Margaret: Collapse of the Lung Associated With Primary Tuberculous Lesions, Thorax **5**: 1, 1950.
13. Baldry, P. E.: Carcinoma of the Middle Lobe, Thorax **7**: 240, 1952.
14. Valle, A. R., and Cloutier, W. A.: Middle Lobe Syndrome Produced by Foreign Body in Lower Third of Esophagus, J. A. M. A. **152**: 812, 1953.
15. Wohl, G. T., Israel, H. L., and Frobese, A. S.: The Middle Lobe Syndrome and Histoplasmosis, J. Einstein Med. Cen. **1**: 17, 1952.
16. Puckett, T. F.: Pulmonary Histoplasmosis. A Study of Twenty-two Cases With Identification of H. Capsulatum in Resected Lesions, Am. Rev. Tuberc. **67**: 453, 1953.

LEIOMYOMA OF THE ESOPHAGUS

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THE first report of an esophageal tumor presumed to originate from muscle was made by Monro in (1797);[59] however, he failed to furnish histologic evidence thereof. Such was also the case in the similar reports of Bell in 1816, Lippich in 1838, Meyer 1882,[53] and Kraus in 1902.

In 1863, Virchow[83] first recorded the anatomopathological characteristics of a leiomyoma of the esophagus. Numerous other case reports followed, but were almost all post-mortem findings.

In 1932, Bezza[6] reported five personal cases and reviewed thirty-nine from the literature. He concluded that esophageal myomas were very rare and of little practical importance.

In the last twenty years there has been a progressive increase in the recognition of these tumors. In this period the reports were, for the most part, made not by anatomopathologists, but by surgeons: Ohsawa,[61] Brock,[9] Harrington and Moersch,[38] Calmenson and Clagett,[13] Schafer and Kittle,[74] Kenworthy and Welch,[42] Harrington,[39] Placitelli,[66] Hurwitz,[40] Manzocchi,[52] Lortat-Jacob,[45-47] Chi and Adams,[16] Cornell and co-workers,[19] Daniel and Williams,[21] Dugan and Meagher,[23] Rossello,[71] Stojanovic and Jelijievic, Goldman and Masters,[32] Garlock and Stegmaier,[30] and Chalnot and co-workers.[15]

The reports in the literature on leiomyoma of the esophagus can be divided into three periods: an early one, from Monro (1797),[59] to Anitschkow (1911),[3] which includes findings of a principally morphologic character; a second period up to Bezza (1932),[6] when investigations were chiefly directed to histogenesis, and a third or contemporary period since Ohsawa's first operation (1933),[61] during which surgeons have treated the problem from a diagnostic and therapeutic point of view.

Frequency.—It seems probable that the frequency of these tumors is not so limited as is generally believed, particularly since certain numbers of cases escape diagnostic confirmation, owing to their lack of symptoms.*

The general incidence of leiomyoma in the alimentary canal, which was established by Smith in 1937, Golden and Stout in 1941, and Oberhelman and co-workers in 1952,[60] puts the greatest number of cases at the level of the stomach and the small intestine. The lowest percentage is met with, according to Smith, at the level of the colon, while in the opinion of Golden and Stout, and Oberhelman and co-workers, it is encountered at the level of the esophagus (Table I).

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*In a very recent publication (J. THORACIC SURG. 27: 1, 1954), Richard H. Sweet reports on 20 cases of benign tumors of the esophagus, of which 17 were classified as "leiomyomas."

TABLE I. SUMMARIES OF TOTAL REPORTED CASES OF LEIOMYOMA

AUTHOR	YEAR	TOTAL NO. CASES REPORTED	LOCATION			
			ESOPHAGUS	STOMACH	SMALL INTESTINE	COLON
Smith	1937	513	50	321	109	33
Golden and Stout	1941	975	51	629	197	98
Oberhelman, Condon, and Guzauskas	1951	1105	66	705	225	109

In regard to the relative frequency of leiomyoma of the esophagus, it has not been possible to establish an exact and complete comparison, either with dysphagias in general, or with the other benign and malignant neoplastic forms of the esophagus. Such a comparison has been made by only a few authors, and most of the cases in the literature have been reported as isolated instances, without any reference to the absolute or relative frequency of the tumors. Of 4,000 patients with dysphagia, Vinson, Moore, and Boromy, of the Mayo Clinic, found three benign esophageal tumors, while Moersch and Harrington reported 15 among 11,000 cases of dysphagia. These last two authors, surveying a series of 7,459 autopsies at the Mayo Clinic, noted 44 benign tumors of the esophagus, of which 32 were leiomyomas. Chi and Adams have reported a list of all the cases of benign esophageal tumors published up to 1948, which showed a 50 per cent greater frequency of esophageal myomas over all other benign neoplasms in the same location. The figure quoted by Calmenson and Clagett (70 per cent) is even higher.

Regarding the frequency of the benign tumors of the esophagus in respect to carcinoma, Chi and Adams reported that in the Clinics of the University of Chicago only two benign tumors of the esophagus were noted, as compared with 246 carcinomas in the same location, with an incidence, therefore, of less than 1 per cent in comparison with the malignant tumors.

Sex.—The sex distribution of the cases in the literature is as follows:

Males	52
Females	26
Sex not identified	22

Bezza attributes such greater incidence to the more frequent causes of irritation to which the esophagus in the male sex is subjected (dietetic disorders, alcoholism, etc). This hypothesis would seem more applicable to tumors which have their origin in the mucosa rather than to those which arise from the muscle.

Age.—Regarding frequency in relation to age, the percentages are as follows:

	Cases
From 11 to 20 years	5
21 30	8
31 40	9
41 50	21
51 60	15
61 70	13
71 80	7
Age not defined	22

From these statistics the deduction might be drawn that leiomyoma of the esophagus is more frequent after the age of 40, as is maintained by some authors,[6, 13] who base their deductions on analogous statistical, albeit numerically inferior, conclusions.

Such conclusions do not seem to be acceptable when dealing with benign tumors of slow development, which are for the most part asymptomatic, at least in the early stages of the disease.

It may be added that the ever-increasing frequency of the neoplasms noted in young patients, possibly thanks to improved diagnostic technique, has drawn some authors, Rossello, for example, to conclude that it is the young who are chiefly subject to such afflictions.

Location.—The cases in the literature are distributed thus:

Leiomyoma of the upper third	14
Leiomyoma of the middle third	23
Leiomyoma of the lower third	37

For gastroesophageal leiomyomas Manzocchi[52] has correctly advocated separate classification, including in such a category the cardial region extending to the gastric wall.

PATHOGENETIC CONSIDERATIONS

The histogenetic problem of these tumors is continually under discussion, but there are three principal theories. The most accredited of these[5, 26] places the origin of the tumor in the esophageal muscle membrane, while Aschoff also suggests that it may originate from the muscularis mucosa, a theory which is, however, excluded by Fischer.[26] To admit such a contingency, according to Rodino' di Miglione,[69] a demonstration of continued affinity between the muscle tissue of the esophagus and that of the tumor is not necessary, however, indispensable serial sections of the entire mass would be. The transition zone can be very circumscribed and limited to one determined sector only.

The second theory, which puts the origin of esophageal leiomyoma in the vessel musculature, has been discussed by many authors, but almost unanimously rejected.[26, 82] Some authors, such as Businco, hold it to be possible, thereby attributing a very rare origin to the esophageal area, especially for those tumors in which vascularization appears to be autonomous and well ordered. Rodino' di Miglione also considers the hypothesis of vascular origin rather forced for such an organ as the esophagus, in which an abundance of muscle tissue already exists. It might be accepted to explain the rise of leiomyomas in organs in which smooth muscle fibers do not normally exist.

The third theory, according to the well-known ideas of Durante and Cohnheim, considers the origin of esophageal muscle tumors, as of analogous neoformations of other parts of the alimentary canal, to be in aberrant nodes of embryonic muscle tissue. Di Falco[22] accepts this hypothesis on the basis of a personal finding of a mediastinal myomatous node, independent of the esophagus and adhering to a lamina of tissue structurally similar to the esophageal wall.

This theory would not oppose the two preceding ones, since the aberrant germ can originate from various layers of the esophageal muscle or from the muscle fibers of the vessels.

PATHOLOGIC ANATOMY

Macroscopic Appearance.—This often varies, depending on how and when the tumor is discovered. In asymptomatic cases, and in incidental post-mortem findings, the tumor, rounded or ovular in shape, has a limited size, rarely larger than a hen's egg.[6, 18, 24, 57, 82] In those cases which have been clinically confirmed, the neoplasm generally presents a more varied appearance, plurilobular in shape like a horseshoe, or a plaque, generally of larger size.

The tumor is firm in consistency, except in a limited number of cases;[27, 82] the outer surface is smooth or rounded. A cross section shows, in almost every case, gray-white, mother-of-pearl color. Sometimes reddish striations are visible, due to better vascularization, or dark brown areas of tissue degeneration,[30] or hard zones of calcification.[3, 6, 41]

According to whether the point of origin corresponds to the muscularis mucosae or is intramucosal, such neoformations can arise in the esophageal lumen and remain intramural, or develop in the periesophageal area.

If it advances toward the esophageal lumen, the tumor is completely or largely covered by mucosa, by the adventitia if it develops toward the outside. Frequently, placed like a capsule, it appears surrounded by connective tissue, which permits discrete mobility. In other cases, it seems to be part and parcel of the esophageal muscle, characterized by a certain immobility, while the mucosa and adventitia remain free and mobile on its surface.

Anitschkow's opinion, according to which only old or very large tumors are capsuliform, is in contrast to the findings of small, slow-growing and distinctly encapsulated leiomyomas of the esophagus, as maintained by Tschlenow, Businco, Rodino' di Miglione, and Garlick and Stegmaier.

The esophageal area above the tumor mass sometimes presents dilation of the lumen and hypertrophy of the muscle, with appearances similar to the "widespread myomatosis" described by Lortat-Jacob.[60]

Classification.—Almost all authors report on benign tumors of the esophagus without following any classification. Some[2, 30, 38, 63, 71] distinguish two categories: tumors originating from the mucosa and submucosa, including fibromas, adenomas and fibroadenomas; and tumors which arise from the rest of the esophageal wall, called by Garlock and Stegmaier extramucosal, the most frequent of which are leiomyomas.

A characteristic of the first group would be pedunculization in the lumen, and of the second intramural location. This affirmation, however, does not always correspond to reality, owing to the existence of leiomyomas possessing a long pedicle and arising in the esophagus.[18, 67, 81]

As far as concerns esophageal leiomyomas, it does not appear to me that an exclusive classification was made in the past in their case.

Recently Rossello has advocated the distribution of cases in the literature into one of the three following groups:

1. Leiomyomas with isolated nodes: This type generally appears as a single node, varying in size from a grain of rice to a walnut, and is usually located at the top of the tracheal bifurcation, or at the cardia. The smallest myomatous nodes observed have been at the cardia.[44, 61] A leiomyoma in isolated form can, even though very rarely, appear at multiple sites.[6, 19, 57, 82]

2. Leiomyomas with multiple nodes: These generally reach the dimensions of an egg, although they are sometimes larger than a fist. Limited to a tract of the esophagus which generally represents the greatest axis of the mass, the tumor appears as if made of multiple myomatous nodes crammed one on top of the other, situated equally in the mid third or lower third of the esophagus. The dimensions of the single nodes generally vary in size from a hazelnut to an almond.

3. Leiomyomatosis or widespread myomatosis: In an area of the esophagus, variable in length, but generally from 5-12 cm., numerous myomatous nodules are to be found in one or more of the muscle layers. These are evident after bisection of the esophageal wall, which itself shows only a greater firmness than usual to palpation.

Sometimes true nodular formations do not exist, mere myomatous zones being included in the thickness of the muscle that are recognizable to careful palpation as a granular and diffuse thickening of the wall, and visible only under the microscope (the external appearance of the esophagus is hardly or not at all modified, and can be said to be normal). To this last category belong Lortat-Jacob's four cases in which the operation (resection of a total longitudinal strip of muscle 2 cm. wide, extending from the aortic arc to the cardia) was carried out because of esophageal dyskinesia or diffuse spasms of the thoracic esophagus.

Such a classification errs perhaps in excessive formalism; it presents, nevertheless, a certain practical utility, since to each of the three groups there often corresponds a particular clinical and radiologic case, and, in consequence, particular therapeutic methods.

Microscopic Appearance.—The characteristic structure of these tumors is represented by little bunches of smooth muscle fibers, which course along without any order; they are located first haphazardly, then longitudinally, now transversely, now obliquely. Vortical placing, characteristic of tumors of an analogous structure in other organs (the uterus, for example), has not been noted in these leiomyomas.

The fibrocellular nuclei, narrow and long, are rounded at the ends. Ovular, rounded, or spindlelike shapes, however, are not unknown.[6]

The protoplasm may appear smooth, homogeneous, or with extremely fine striations (Bezza was able in one case to distinguish the myofibrils).

The connective tissue is sometimes particularly ample. We speak in such cases of leiomyoma fibrosum or of fibroleiomyoma; often the little muscle bunches have very fine connective fibers permeating them, and connective elements are at times present between the individual muscle cells.[3, 11] According to Bezza,[6] a net, which is collagenous in nature, exists effectively around each single fiber, but a connective tissue would be present between cells only in particular conditions. Abundant elastic fibers are sometimes attached to some myomatous tumors, especially if pedunculated. The vascular element is generally scarce; vascular lacunae are often recognizable rather than true and proper vessels. Bezza, in his five cases, found fissures with an endothelial wall, lacking red

corpuscles, very probably interpretable as lymphatic lacunae. We can often observe degenerative and necrotic phenomena, and calcareous deposits visible sometimes even to the naked eye.

In "widespread leiomyomatosis,"[745] the nodules (more or less distinguishable) are contiguous and appear in rosary form. Composed of muscle cells of variable thickness and size, which intersect each other with order, they frequently reveal an interstitial infiltration of an inflammatory nature.

Fig. 1.

Fig. 2.

Fig. 1.—Case 1. Preoperative esophagram demonstrating displacement to the right in lower third of esophagus and concave appearance of its left wall. The arrows indicate the tenuous rounded shadow of the tumor mass.

Fig. 2.—Another esophagram of the same case.

CASE REPORTS

CASE 1.—M. P., Aged 54 years, was hospitalized June 11, 1949.

Operative diagnosis: Tumor of the esophagus (lower third).

Case history: One year prior to admission the patient suffered morning attacks of vomiting. Three years prior to admission he had had a similar vomiting episode of ten days' duration. Roentgenogram examination revealed minimal esophageal stenosis. From July to October, 1948, he underwent medical and roentgen therapy in the Montreal Hospital. No weight loss was ever noted.

System review: Physical and laboratory examinations were normal.

Respiratory system: There was nothing abnormal in the chest. Clear plexus sound was heard.

Radiologic examination of the alimentary canal: Esophagus was slightly enlarged. In its lower part, where evident mucous changes were noted in addition to irregularity and rigidity in its left outline which showed a slightly concave appearance as from extrinsic pressure, it seemed to have moved to the right and back. In this location there was a tenuous

shadow, well-defined laterally (medially it was confluent with the esophageal wall on which it would seem to have attached itself), and which could be ascribed to a tumorous process. The opaque passage through the terminal esophagus was not sensibly obstructed, even though a slight bulge was noted above.

Conclusions: There were organic changes in character of the terminal esophagus, ascribable to a neoplastic process in the left lateral wall; development chiefly external, but also intramural. Esophageal lumen adequate.

Operation (By Professor Forni, March 13, 1949): Endotracheal general anesthesia was administered. Thoracophrenolaparotomy (subtotal resection of the seventh rib) was performed showing total pleural and partial sternopericardiac symphysis, which were freed.

Fig. 3.

Fig. 4.

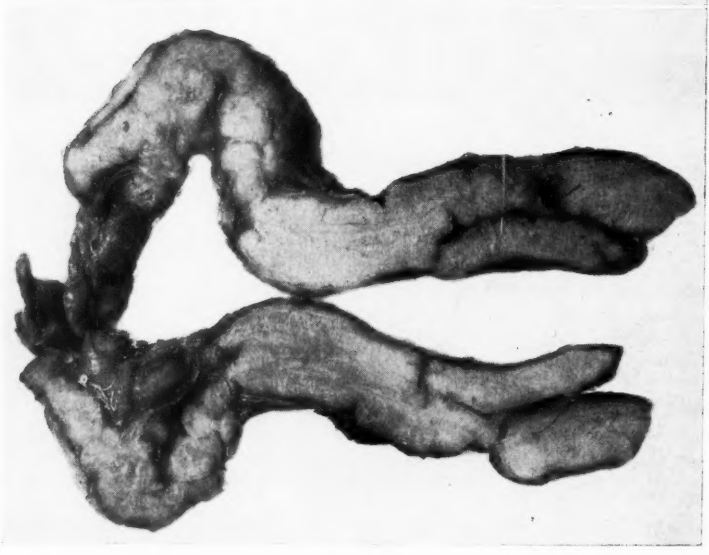

Fig. 3.—Case 1. External appearance of the tumor, sinuous in form, with characteristic lobulation.

Fig. 4.—Case 1. Section of the tumor.

Fig. 5.

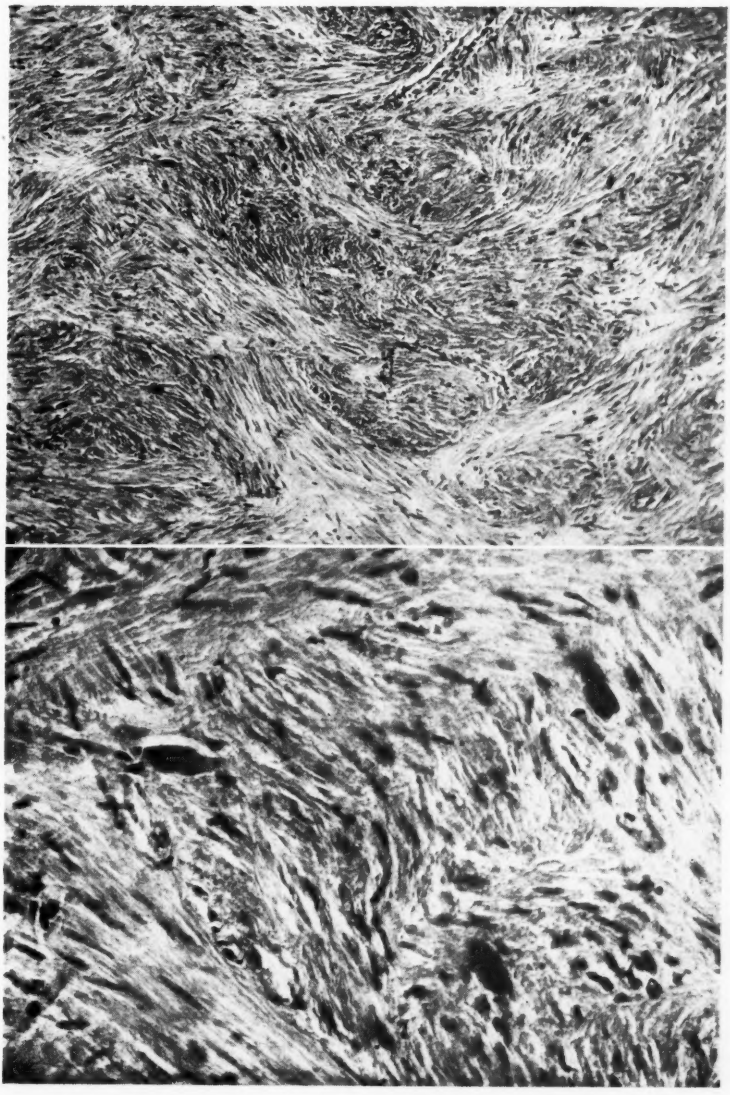

Fig. 6.

Fig. 5.—Case 1. Photomicrograph of the tumor, demonstrating various groupings of muscle cells affected by incision. (Van Gieson stain; ×67.)

Fig. 6.—Case 1. High-power photomicrograph of the tumor (×395); of the outstanding phenomena, the degenerative ones are particularly noticeable.

The diaphragm was incised and the esophagus mobilized, at which point a tumor, folded in U-form, was found in the wall thickness, which bulged considerably from under the adventitia. The adventitia was incised, and the procedure for isolating the tumor, which was about 9 cm. in length, initiated. During the enucleation process the mucosa was opened at two limited points. The tumor appeared to be of an irregular spindlelike form, with the U-shaped folding described; it was elastic in consistency, yellowish-white in color, lobulated, and probably of connective tissue structure. Triple layer closure of the esophagus was done, thorax was closed without drainage, penicillin was used endopleurally and intramurally.

Fig. 7.

Fig. 8.

Fig. 7.—Case 1. Postoperative esophagram six months after; demonstrating good esophageal canalization.

Fig. 8.—Case 1. Postoperative esophagram twelve months after, demonstrating the continuance of the esophagus in good working order.

Histologic report: The tumor was composed of tissue, compact in structure, and bunched in type. Such tissue appeared sometimes richer, sometimes poorer, in cellular elements, disposed in rectilinear or undulating bundles, variously placed, now diverging, now intersecting. The stroma and the scarce intercellular substance were composed of delicate collagenous homogeneous fibrils, with here and there a slightly hyaline appearance. Vessels, mostly very small in size, were running through this stroma and presented almost one endothelial wall only; they seemed to be intimately interwoven into the tissue, with some lymphomonocytic element in the adventitia.

The cellular elements which composed the tumorous tissue did not present a uniform structure. For the most part it proved to be of greatly elongated cells, whose cytoplasm, slightly eosinophilous, had somewhat uncertain limits, and was characterized by a certain longitudinal fibrillar disposition, disappearing at the poles. The nucleus was slender in form, at times a little curved as a comma, threadlike or tortuous. Larger ovular nuclei were sometimes seen with the chromatin in a hyperchromic homogeneous manner; the limits were not sharp and gave the impression of a swollen and worn tissue. For the most part such appear-

ances coincided with the regressive phenomena of the cytoplasm and with the formation of cytoplasmatic lumps. Other elements were more intimately interwoven in the collagenous tissue.

With the exception of the mentioned appearance (sometimes gigantic) of nuclear regression, neither karyokinesis nor signs of abnormal proliferation were noted.

A trichrome stain (Masson, Van Gieson) showed the muscular nature of the tumor distinctly.

Histologic diagnosis: Leiomyoma.

Postoperative course: The patient had an uneventful convalescence. After two weeks the wound was well healed and the patient was eating without difficulty. Barium swallow revealed a satisfactory anastomosis. The patient was discharged March 30, 1949, and continued his normal life in Canada completely free of any complaints. He died three years later from cardiac insufficiency and acute concomitant pulmonary affection.

This case offers some interesting clinical and histopathologic consideration. Of particular interest was the prolonged symptomatology, which was, in fact, very unusual and limited to vomiting attacks in the morning prior to eating, without other characteristic signs of esophageal stenosis. The tumor mass, as shown in the roentgenogram, was at first considered to be of a malignant nature, so much so as to warrant a course of roentgen therapy. The prolonged symptoms, their lack of progression, the unchanged body weight, the good general condition, and an esophageal lumen clinically and radiologically maintained suggested, however, a benign process confirmed by the operation and histologic report.

The prominent regressive phenomena, which characterized the microscopic findings, and which have already been described by other authors, are worthy of note. Ribbert found on the cell periphery the presence of hyaline bodies which he considered to be parasites. Houette excluded such a hypothesis, but maintained the little hyaline lumps were caused by precipitation of the protoplasm for changed colloidal equilibrium of the cellular protoplasm. More recently, Bezza declared such appearances to be regressive phenomena, in the sense of myofibril fragmentation and decomposition.

In our case and in contrast to the findings of other authors, the degenerative phenomena chose to affect the nucleus, probably as a result of the roentgen therapy carried out six months prior to the operation. Such a finding might acquire a notable significance, since descriptions of leiomyomas enucleated after roentgen therapy have not been made.

CASE 2.—Z. E., aged 21 years, was hospitalized March 4, 1953.

Preoperative diagnosis: Tumor of the esophagus (lower third).

Case history: The tumor in question was discovered by chance in the course of a recent roentgen examination one month prior to hospitalization, necessitated as a result of about a three-month persistence of slight asthenia, lack of appetite, postprandial epigastric swelling, and pyrosis. The patient attributed these sensations, which had appeared in previous years in a mild form and for brief periods, to his particular type of work (in a factory where the air was saturated with dust and chemical vapors) and to the pressure of the shoes on his epigastrium while working on the lathe.

The patient experienced no other disturbances—no dysphagia, no regurgitation, and no pain.

Physical examination: System review and laboratory studies were negative.

Radiologic examination of the alimentary canal: Esophagus was slightly dilated, especially in its lower part, where two lacunary forms were noted about the size of a hazelnut, and located in the epicardial site. The mucous folds were soft, and the opaque passage into the stomach was not obstructed.

The stomach, of hook form, was normal in size, empty prior to eating, and without organic forms. There was complete peristalsis on both curvatures. Duodenal bulb was regular. Second portion was normal in appearance. Time of emptying of the stomach was within physiologic limits. Passage through small bowel was normal.

Conclusions: Intraesophageal neoplastic process affecting the epicardial section, polypoid in type.

Esophagoscopy: Esophagus was normal in form, size, and consistence as far as mid third. Lower third appeared slightly dilated with the mucosa stretched out. In the area immediately above the cardia two roughly-rounded formations, which were pendulous, completely covered by mucus and not bleeding, were observed. One of these was situated on the left on the anteriolateral aspect, the other slightly lower on the right, on the posterolateral. Firm in consistency, not very elastic, they did not obstruct the passage. Diaphragmatic movements lifted the two formations synchronously.

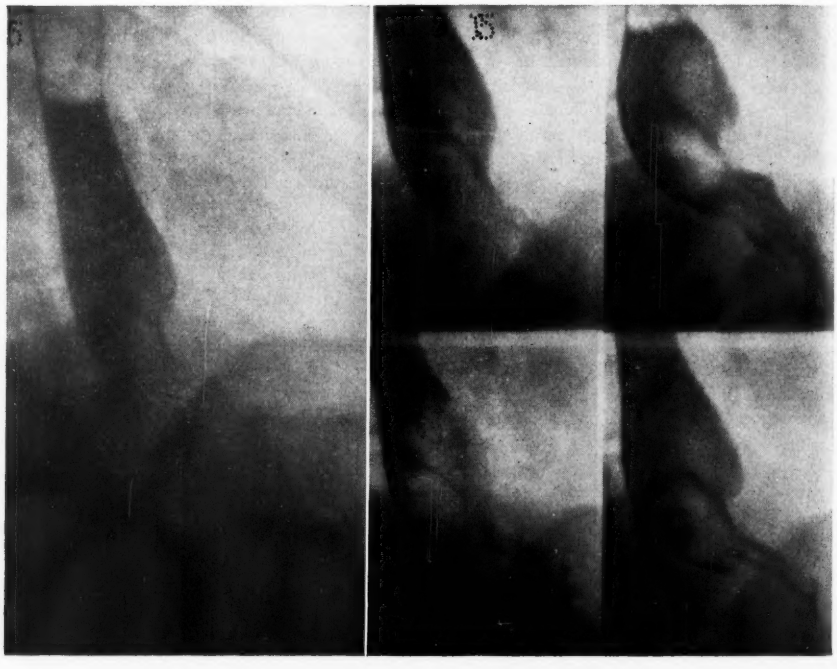

Fig. 9. Fig. 10.

Fig. 9.—Case 2. Preoperative esophagram demonstrating dilation in lower third of esophagus, the presence of two lacunary images and the regularity of the mucous folds.
Fig. 10.—Case 2. Another esophagram of the same case.

Operation (By Professor Forni, March 28, 1953): Thoracophrenolaparotomy was performed. By incising the mediastinal pleura and dissecting the triangular ligament, the esophagus was taken out through the hiatus. It was isolated for an area of about 10 cm. In the wall a slightly knobby neoformation presented itself, which extended about 7 cm. in length. At the cardia it took on a U shape and then rose into the opposite wall in a space of about 4 cm., taking on a squat, but ever-knobby appearance. The muscle wall was incised, and the shelling-out of the tumor, which was surrounded by a small layer of adventitia, was begun. The process of enucleation was carried out without entering the mucosa except in an area of some millimeters, where the apex of the tumor joined with the mucosa, which was closed with a suture. The finding was analogous to that in the preceding case in regard to shape, size, and appearance.

Histologic report: The fragments, taken at various points from the little mass, presented a bunched structure throughout. The tumor consisted of currents of cellular elements, variously set, greatly elongated, some of which were disposed in a parallel and, at

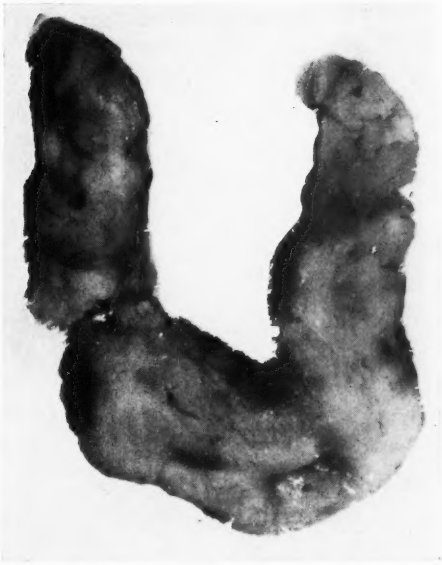

Fig. 11.—Case 2. External appearance of the tumor, showing lobulation and U shape.

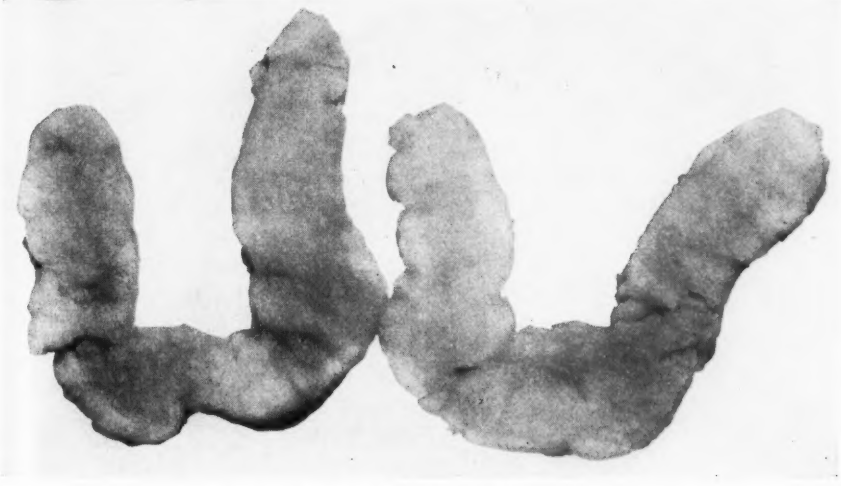

Fig. 12.—Case 2. Section of the tumor.

Fig. 13.

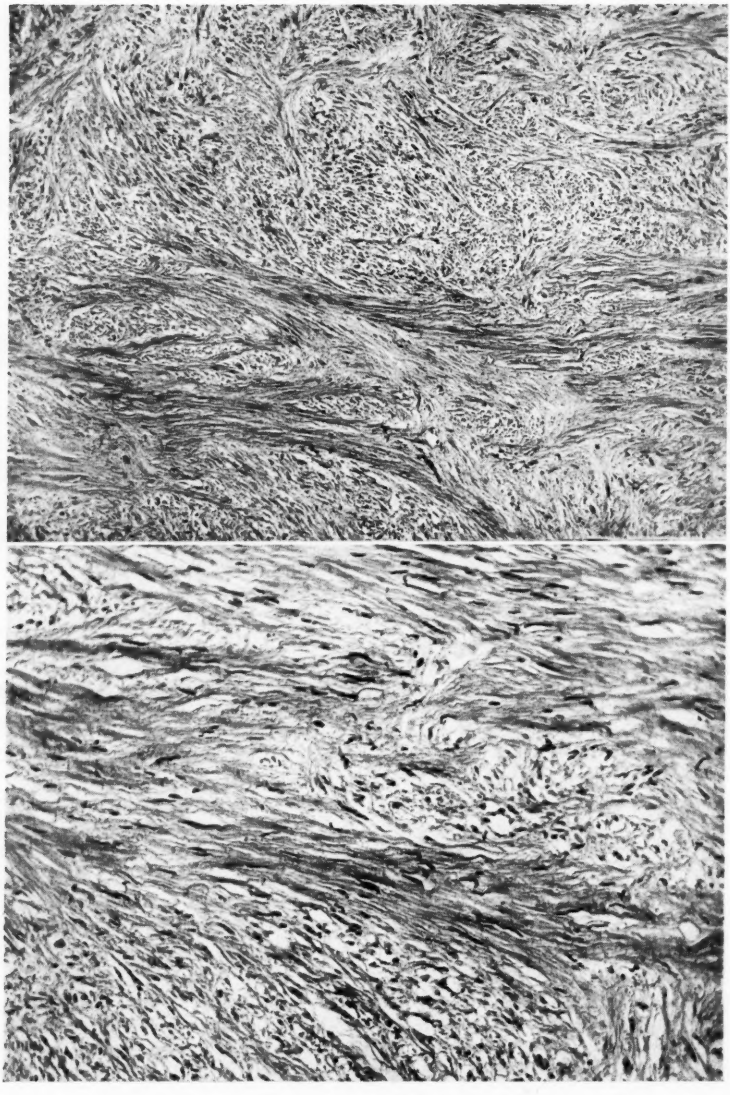

Fig. 14.

Fig. 13.—Case 2. Bunched structure of the tumor, determined by currents of fibrous muscle cells variously situated (×80).

Fig. 14.—Case 2.—Enlarged detail of Fig. 13, showing the longitudinal striation and the spindle- or threadlike appearance of the nuclei (×319).

the same time, sinuous manner. Sometimes too, these cells were grouped in large vortices, so that the microscopic section caught the cell bundles in various incidental groupings.

Some fine fibrillar septa united the bundles, which were particularly dense around the vessels, losing themselves in the muscle wall of the latter.

The cytoplasm also was sometimes not clearly distinct from the fibrillar stroma. Where it was better preserved, it was possible to distinguish a longtitudinally-striated structure and a considerable eosinophilia, with some swelling here and there. The differentiation between the cellular elements and the stroma, which appeared otherwise run together, made itself particularly evident with the elective staining (Mallory, Van Gieson).

The nuclei were spindlelike, sometimes curved in common form, sometimes somewhat undulating or stretched out in threadlike appearance. The chromatin content of such nuclei was regular and finely powdered, with some little nucleoli here and there.

Histologic diagnosis: Leiomyoma.

Postoperative course: The postoperative course was uneventful. The patient was eating a full diet on the tenth postoperative day and had a well-healed wound.

In this patient too, the symptoms, although minimal, had been in existence for some years, characterized by long periods of remission and by a complete absence of dysphagia, regurgitation, or pain. The only sensations were moderate asthenia, lack of appetite, and at times postprandial epigastric swelling and pyrosis.

Esophagoscopy and roentgenograms demonstrated the existence of the tumor, the prevalent development within the lumen and polypoid characteristics, the mucous integrity, the probable benign nature of the tumor, and its probable origin in the muscle wall. The operation confirmed such impressions, producing a tumor with characteristics analogous to those in Case 1, but with a tendency to development toward the esophageal lumen, as to distend and notably thin the mucosa, and to take on at some points a polypoid appearance.

The histologic examination demonstrated the same bunched structure, as in the preceding tumor, and the same cytoplastic and stromal characteristics, but showed complete absence of any aspect of nuclear regression.

CLINICAL COMMENTS

Esophageal myomas are frequently asymptomatic. Of 95 cases in the literature, 61 were free of clinical symptoms.

The appearance of symptoms is sometimes preceded by badly defined disturbances, or painful sensations, which the patient is often unable to describe, or which are sometimes considered to be of a functional nature, as by Lanzara.[44]

Dysphagia is a very frequent isolated symptom,[3, 18, 44, 49, 61, 71, 81] but more often accompanied by other disturbances. It is generally found with ingestion of solid foods, and rarely with liquids. In the case referred to by Lanzara,[44] the patient, to feed himself, was obliged to recur to Cohn's maneuver (rapid swallowing of a glass of water after every mouthful of food). Sometimes dysphagia accompanies only certain foods, as in Cornell and Shehady's patient[19] (dysphagia with sweet, cold, or acid foods). Very often it appears at irregular intervals, with more or less rapid regression. It rarely shows a course constant or progressive to the point of stenosis.[3, 18, 45, 66] It can persist for years, as follows: twenty-four years,[61] seven years,[30, 50] and six years.[44]

Schafer and Kittle[74] hold that benign tumors causing dysphagia are rarely extramucosal in origin (myomas), but that their origin is more often submucosal or mucosal, since these last pedunculate easily, intruding into the esophageal lumen. Dysphagia secondary to leiomyoma, according to the same authors, should be due either to great enlargement or to malignant transformation. From the literature, however, it is inferred that dysphagia is present also in small-sized tumors.[3, 30, 44, 61, 70]

Pain during deglutition, particularly of solid food, has been described only by Garlock.[30] Frequently the pain is, instead, spontaneous, or begins immediately,[71] or about ten minutes after eating.[37] It is more frequently retrosternal,[30, 34, 39, 71] but can also be found in the thorax,[52, 74] in the epigastrium,[23, 52] and even in the shoulders.[30] It can be continuous, but more frequently is crampy with spontaneous remissions and exacerbations.

Sometimes nausea and vomiting[23, 45] are present, or direct alimentary vomiting takes place.[9, 30, 37, 71, 74] Other effects are regurgitation,[30, 44, 45] a sensation of weight in the epigastric region,[9, 19, 37] and dyspeptic sensations, in which periodic remission of the symptoms makes one suspect the presence of an ulcer.[45] No author has found sialorrhea.

Sometimes there can be loss of weight, depending on the grade and duration of the dysphagia. If this regresses, the recovery of body weight can be evident in a short time.[45] The general state of health is, however, generally well maintained, even many years after the beginning of the illness.

There have also been descriptions of symptoms of compression of near-by organs (trachea and bronchi) with dyspneic crises[62] which, nevertheless, might be independent of stenosis. Dyspnea associated with coughing is present in Manzocchi's case.[52] (Leiomyoma of the lower third of the esophagus came to resemble an irritation of the vagus nerve.)

The fundamental characteristic of the symptoms explained is their notable duration—sometimes for years—and their capricious evolution.

In two patient[45] hemorrhage, which showed itself with hematemesis and melena, was experienced. Lippich described perforation of the trachea by a tumor, whose muscular nature was not, however, histologically documented.

DIAGNOSIS

In leiomyomas of the esophagus a positive clinical diagnosis is not possible. In fact, dysphagia, retrosternal or epigastric pain, the sensation of weight, nausea, and vomiting are not characteristic, but common to all tumors of the esophagus and periesophageal spaces.

A diagnosis of probability is possible,[19, 23, 37, 38, 45, 71] and, in our second case, in addition to an accurate differential diagnosis by x-ray examination and esophagoscopy, completed, if necessary, by biopsy. This last is, however, not always advisable, because of the necessity of penetrating the integral mucosa to reach the tumor and because of the possibility of creating wounds and trauma, which might prove serious.

The radiographic image of these neoplasms presents appearances different in accordance with their anatomic form.

In the rare cases of pedunculated leiomyoma, one can perceive a particular shadow which cuts into the esophageal lumen and which coexists generally with a wall which is functionally intact.

In the intramural forms or in those where the tumor arises in the periesophageal space, a simple roentgenogram can demonstrate the presence of a rounded shadow, more or less opaque, with clean edges, and well defined in respect to the surrounding tissue. In the barium swallow, the tumor mass is confused with the esophageal wall. There often exists, at the same time, dilation

and deviation of the esophagus, whose lumen generally presents deformations (Case 1). The opaque meal passage is not sensibly obstructed, although a slight stoppage above is to be noted.

Harper and Tiscenco[37] tried to enumerate the most important radiologic characteristics, which are useful mainly in the differential diagnosis with carcinoma of the same site. The authors underlined the importance of a tumor mass, for the most part not fixed, connected with the deformed esophageal area and more or less swollen in the mediastinum. They recognized the frequent enlargement at the same level of the tumor, or a protrusion of that part of the wall on a level with and opposite to, the tumor; the absence of undermined margins, the variations in the form and size of the refilling defect, the absence of any ulceration of the mucosa corresponding to the neoplasm, and the conservation of the normal fold in the immediate neighborhood of the refilling defect.

Recently Chalnot and co-workers[15] underlined the importance, after a barium swallow, of some aspects of the lacunar image, which, characterized by limits more or less regular, frequently reproduces lobulation typical of leiomyomas covered by integral mucosa and presents at its extremities two little spurs in relation with the superior and inferior poles. Such signs indicate the intactness and softness of the mucosa and are opposed "to the rigid, fragmentary, and irregular appearance of the mucosa affected by a malignant neoplasm."

Lortat-Jacob[45] noted a kind of arborescent image, in a leiomyoma with a macroscopic appearance similar to our first case, without amputations, due, in his opinion, to the barium filling up the crevices provoked by the lobulations of the tumor. The same author has also reported a flattening of the esophageal profile, which reminds one of ureterographical images for a ureter flattened like a sword blade. Such an appearance would be due to the particular and frequently noted horseshoe form of the tumor.

Of particular moment in the two personal cases are the enlargement and the displacement of the area of the esophagus affected by the tumor, the presence of concavity as a result of extrinsic pressure, the existence of a regular-rounded shadow, the uniformity of the mucous layout, and the complete absence of obstruction in the barium passage.

A careful analysis of the radiologic findings (before or after barium meal), especially if combined as a result of repeated tests, can permit within a limited field of error not only the generic diagnosis of a benign tumor of the esophagus, but the ascertainment of the eventual preponderant development (intrinsic or extrinsic) assumed by the tumor mass.

Esophagoscopy produces good results in pedunculated tumors, whose size, shape, location, and origin can be evaluated with certainty.

In intramural leiomyomas, or in those which develop in the periesophageal area, an intact mucosa is noted, more or less raised by the underlying tumor. Sometimes the mucosa appears congested[45] or superficially eroded.[30] The esophageal lumen at the level of the tumor can be restricted so as not to permit the passage of the esophagoscope[19]; in the overlying esophageal area, dilatation, usually in a limited degree, is frequent.[44, 45]

During esophagoscopy, it is sometimes possible to take up a fragment of tissue for a histologic examination which alone can permit a definite diagnosis. We have previously hinted at the obstacles to this procedure, and at the necessity of preserving intact the mucosa, which is often extended and very friable.

DIFFERENTIAL DIAGNOSIS

Of the tumors which recur frequently in the differential diagnosis with leiomyoma of the esophagus, let us consider above all carcinoma, sarcoma, the other benign neoplasms, megaesophagus, and cardiospasm—in short, the neoplasms and malfunctions located in the periesophageal area and compressing the esophagus.

Carcinoma in general presents a symptomatology briefer in duration and an evolution which is progressively fatal. Esophagoscopy shows, in the ulcerated forms, the loss of substance and edges slightly raised, and in the vegetant ones a polypoid or fungus appearance, a notably hard bulge in the infiltrating types, which tends to compress the lumen. Bleeding, moreover, is easy.

Sarcoma is more often characterized by a rapid evolution, the absence of linear infiltration frequent in the epithelium, and the not infrequent pulmonary, pleuromediastinal, and tracheobronchial metastases.

The differential diagnosis, based above all on the sometimes prevalent copious esophageal dilation above the leiomyoma, is also valid for esophagospasm and megaesophagus.[44, 45]

For the diagnosis of cardiospasm x-ray findings are necessary of the image of the esophagus in cigar form or of the cardiac channel as a root or rat's tail, and esophagoscopic images with an obstruction in the passage of the instrument which proceeds slowly but completely.

Abundant regurgitation, a postprandial sensation of obstruction which passes sometimes suddenly with a special noise, dysphagia, and retrosternal oppression which tend to increase progressively, bad oral taste, and the characteristic sensation of entry in the cavity of the esophagoscope, once cricoid restriction has been overcome, incline one toward the diagnosis of megaesophagus. The diagnosis is confirmed by the radiologic examination, which shows an esophagus notably dilated in sack form, with pockets, or cylindrical in shape, in addition to being considerably elongated and sinuous, and the constant integrity of its subdiaphragmatic portion.

In so far as concerns the extraesophageal compressions, the tumors which can recur in the differential diagnosis are listed in a simple manner for purposes of brevity: (1) aneurysm of the aortic arch in its posterior position and of the descending aorta; (2) aneurysm of the left subclavicular artery; (3) bronchiogenic carcinoma, especially of the left main bronchus; (4) cystic tumor, or parasitical (echinococcus) or congenital (dermoid) cysts of the mediastinum; (5) thyroid or retrosternal parathyroid tumors; (6) lymphoglandular tumefactions (lymphosarcoma, lymphogranuloma); (7) folded esophageal diverticulum, modified in its radiologic appearance by state of repletion (saliva, food); (8) diaphragmatic hernia.

THERAPY

The therapy of leiomyoma of the esophagus is exclusively surgical. Monro[59] and Dubois have carried out a ligature of the pedicle of an esophageal tumor arising into the mouth. In the rare eventuality of a pedunculated leiomyoma, its removal can be carried out orally during esophagoscopy. Almost always, however, the leiomyoma (intramural or subadventitial) requires thoracotomy, generally from the left, but also from the right (Rossello's and Chalnot's cases were localized in the higher mid-third). The method known as thoracolaparotomy,[13, 15, 16, 35, 37, 45, 61, 74, 85, 86, 87, 89, 90, 91, 93] is used if the tumor is located in the lower third or if it affects the cardia.

The esophagus having been exposed, the tumor is seen in its characteristic appearance. Attachments with the near-by organs and the mediastinal pleura, infiltration, sclerosis, or wall retraction, as frequently seen in malignant tumors, do not generally exist.

The neoplastic mass, sometimes lobulated with serpentine prolongations which are more often rounded, appears in strict connection with the muscle membrane of the esophageal wall, and is, for the most part, well enucleable.

The method, followed by a number of workers,[13, 15, 16, 35, 37, 45, 61, 74] consists in a prudent and progressive enucleation of the tumor without entering the mucosa. The mucosa can accidentally be opened in some limited points; however, this has no great importance.

Enucleation is the operation of choice for single-node leiomyomas, but can also be performed for those with multiple nodes when good possibilities of leaving the mucosa intact exist. It presents no particular surgical difficulties or risks, and permits the esophagus to maintain its normal physiologic appearance.[15]

Those leiomyomas which cannot easily be enucleated by reason of their strict continuity with the muscle of the esophageal wall, or because they are intimately attached to the mucosa and those of the cardioesophageal region (which frequently also affect the gastric wall), may require esophageal or gastroesophageal resection, according to the case in question.[9, 32, 38, 42, 45, 52, 66, 71, 76, 78] This is followed, respectively, by end-to-end esophageal esophagostomy or gastroesophageal anastomosis.

Only three patients have died in the postoperative period after such a course of action. The long-term results, in general, have been satisfactory in the repeated control tests, with a good recovery of esophageal canalization. Some rare and temporary spasms may, however, persist, or peptic esophagitis may show itself, as in Lortat-Jacob's patient.[50] Resection is accompanied by greater risks and sometimes by complications in gastroesophageal canalization, considerations which merit accurate evaluation when one is dealing with benign tumors which should require, only in cases of necessity, operations other than simple enucleation.

CONCLUSIONS

Leiomyoma of the esophagus is a tumor which is well defined from the anatomopathological and clinical points of view, notwithstanding its rather rare appearance (102 cases from 1797 to 1954).

It is most frequently found in the lower third of the esophagus. It more often affects the male sex (66.6 per cent) after the age of 50 years. Esophageal leiomyomas in the young are not rare.

According to the best opinions, the tumor originates from the muscle layers of the esophageal wall.

The most frequent anatomic form to appear in autopsy cases has been that of a single node, while in clinical cases the multinodular form has predominated.

Symptoms are common to the tumors which narrow the esophageal lumen. Generally they are of long duration, but without affecting the general well-being —a characteristic moreover, of all benign neoplasms.

Since diagnosis is not always possible merely by evaluation of the symptoms, the importance of such diagnostic aids as radiology, esophagoscopy, and sometimes biopsy, must be emphasized.

The clinical course is, for the most part, slow. Evident findings of malignant transformations have only once been mentioned.

The only efficacious treatment at our disposal is surgical (most frequently enucleation of the tumor without opening the esophageal mucosa), a therapy that leads to really satisfactory immediate and long-term results.

SUMMARY

Two case reports of leiomyoma in the lower third of the esophagus are presented in men of 54 and 21 years, respectively, operated on in the Surgical Clinic of Bologna.

The enucleation of the tumors was followed by recovery, controlled by repeated radiologic examinations (good esophageal functioning).

Such experiences and a survey of the literature on the subject lead to a description of the principal anatomopathological, and clinical characteristics of leiomyoma of the esophagus, with therapeutic and prognostic deductions.

REFERENCES

1. Abraud, L.: Tumeurs de l'oesophage et tumeurs benignes en particuliers, Rev. méd. Paris **21**: 143-149, 1911.
2. Adams, R., and Hoover, W. B.: Benign Tumors of the Esophagus: Report of three Cases, J. THORACIC SURG. **14**: 279-286, 1945.
3. Anitschkow: Ueber Mioma des Oesophagus und der Cardia, Virchows Arch. **205**: 1911.
4. Bell, C.: Surgical Observations Being a Quarterly; Report of Cases in Surgery, London, 1816, p. 77.
5. Berazzuol, E.: Contributo allo studio dei tumori muscolari dell'esofago, Arch. It. Mal. App. Dig. **9**: 41-50, 1940.
6. Bezza, P.: I miomi dell'esofago, Pathologica **24**: 71-84, 1932.
7. Bordone, S.: Sui leiomiomi dell'esofago; studio anatomo-clinico, Arch. It. Otol. **59**: 573-581, 1948.
8. Borst, M.: Tumori del tessuto muscolare e nervoso; in Istologia Patologica. Zanichelli Ed., Bologna, 1944, pp. 354-359.
9. Brock, R. C.: Cardio-Esophageal Resection for Tumor of the Cardia: Report of a Successful Case, Brit. J. Surg. **30**: 146-150, 1942.
10. Bryant, J.: Myoma of the Esophagus, J. A. M. A. **45**: 2008-2010, 1905.
11. Businco, A.: Sui miomi esofagei, Scritti in Onore di R. Silvestrini, An. Fac. med. Chir. Univ. Perugia. **37**: 1-10, 1938.
12. Caliceti, P.: Malattie dell'esofago in Compendio di otorinolaringoiatria, Cappelli Ed. Bologna, 1949, pp. 609-654.
13. Calmenson, M., and Clagett, O. T.: Surgical Removal of Leiomyomas of the Esophagus, Am. J. Surg. **72**: 745-747, 1946.
14. Cavalcabo, G.: Miomi maligni, Tumori **29**: 267-286, 1943.

15. Chalnot, Faivre, Lochard, and Hahn: Les léiomyomes de l'oesophage et leur traitement par enucléation simple, Presse méd. **59**: 224-226, 1951.
16. Chi, P. S. H., and Adams, W. E.: Benign Tumors of the Esophagus; Report of Leiomyoma, A. M. A. Arch. Surg. **60**: 92-101, 1950.
17. Churchill, E. D.: Leiomyoma of Esophagus, New England J. Med. **236**: 955-958, 1937.
18. Coates, J.: Large Polypoid Myoma of the Oesophagus, Glasgow M. J. **4**: 201-206, 1872.
19. Cornell, N., Shehadi, W., and Sharnoff, J.: Leiomyomas of the Esophagus. A Report of Five Cases, Surgery **28**: 881-886, 1950.
20. Cummings, G. O.: Diseases of Esophagus: One Hundred Cases. J. Maine M. A. **37**: 275-287, 1946.
21. Daniel, R. A., and Williams, R. B.: Leiomyoma of the Esophagus, J. THORACIC SURG. **19**: 800-805, 1950.
22. Di Falco, G.: Miomi mediastinici extra-esofagei (contributo anatomo-Patologico). Pathologica **31**: 381-384, 1939.
23. Dugan, D. J., and Meagher, T. H.: Esophageal Leiomyomas. Report of Additional Case, A. M. A. Arch. Surg. **61**: 1066-1073, 1950.
24. Eberth, C. Y.: Grosses Myoma des Oesophagus, Virchows Arch. **43**: 137, 1868.
25. Ferrari, R. C.: Tumores benignos del esofago, Rev. Mex. de Cir. ginec. y cancer **9**: 135-150, 1941.
26. Fischer, W.: Die Geschwülste der Speiseröhre. Handbuch der spez. Path. Anat. u. Hist., Hencke-Lubarsch **4**: 130-132, 1926.
27. Fogge, and Hilton: Myoma of the Oesophagus, Tr. Path. Soc. **26**: 94-96, 1874.
28. Forni, G.: Patologia Chirurgica, Cappelli Ed., 1949, pp. 515-516, Bologna.
29. French, L. R., and Garland, L. H.: Leiomyosarcoma of Esophagus, Am. J. Roentgenol. **45**: 27-31, 1941.
30. Garlock, W. L., and Stegmaier, J. G.: Benign Tumors of the Esophagus; Report of a Case of Leiomyoma, Surgery **29**: 109-116, 1951.
31. Glinski: Ueber Polypenförmige Mischgeschwülsten des Oesophagus, Virchows Arch. **167**: 383, 1902.
32. Goldman, A., and Masters, H.: Leiomyoma of the Esophagus, A. M. A. Arch. Surg. **60**: 559-575, 1950.
33. Gray, H. K., and Sharpe, W. S.: Benign Lesions at the Lower End of the Esophagus, Am. J. Surg., **54**: 252-261, 1941.
34. Grunberger, E. A., and Pijper, A.: A Case of Leiomyoma of the Esophagus, Acta Otolaryng **5**: 26-29, 1923.
35. Guetgemann, A.: Gutartige Geschwülste der Brustspeiseröhre, Chirurg. **3**: 97, 1952.
36. Harper, R. A. K.: Symposium: Non Malignant Conditions of the Esophagus, Brit. J. Radiol. **16**: 111, 1946.
37. Harper, R. A. K., and Tiscenco, E.: Benign Tumor of the Esophagus and its Differential Diagnosis, Brit. J. Radiol. **18**: 99-107, 1945.
38. Harrington, S. W., and Moersch, H. J.: Surgical Treatment and Clinical Manifestations of Benign Tumors of the Esophagus With Report of Seven Cases, J. THORACIC SURG. **13**: 394-414, 1944.
39. Harrington, S. W.: Surgical Treatment of Benign and Secondarily Malignant Tumors of the Esophagus, A. M. A. Arch. Surg. **58**: 646-661, 1949.
40. Hurwitz, A.: Leiomyoma of the Esophagus. Report of a Case, Surgery **25**: 304-306, 1949.
41. Illig, W.: Die Myome des Oesophagus, Inaug. Diss. 1894.
42. Kenworthy, R. A., and Welch, C. S.: Leiomyoma of the Esophagus and Cardia of the Stomach, Surgery **23**: 745-752, 1948.
43. Küttner, H.: Chirurgische Operationslehre, Barth Leipzig **2**: 398, 1934.
44. Lanzara, A.: Leiomioma dell'esofago e sindrome disfagica funzionale, Arch. Chir. **2**: 283-291, 1947.
45. Lortat-Jacob, J. L.: Deux léiomyomes de l'oesophage, Semaine Hôp. Paris **25**: 1709-1713, 1949.
46. Lortat-Jacob, J. L.: Peut-on envisager le démembrement des syndromes fonctionells de l'oesophage? La myomatose diffuse de l'oesophage, Semaine Hôp. Paris **26**: 177-181, 1950.
47. Lortat-Jacob, J. L.: Myomatoses localisées et myomatoses diffuses de l'oesophage, Arch. mal. app. digest **39**: 519-524, 1950.
48. Lortat-Jacob, J. L.: La Myomatose nodulaire diffuse de l'oesophage, Acquisitions Méd. Réc. 1950.
49. Lortat-Jacob, J. L., and Badaro: Deux sarcomes primitifs de l'oesophage pédiculés. Semaine Hôp. Paris **26**: 37, 1950.
50. Lortat-Jacob, J. L.: Monstrueux léiomyome de l'oesophage. Résection. Semaine hôp. Paris **27**: 69, 1951.
51. Maucher, S.: Die Gutartigen Geschwülste des Oesophagus, Inaug. Diss. Zürich, 1900.
52. Manzocchi, L.: Il leiomioma Gastro-esofageo. Chirurgia **4**: 349-360, 1949.
53. Meyer, J.: Ueber Einen Fall von Miom des Oesophagus, Inaug. Diss. München, 1882.
54. Meyer, Ph.: Ueber Knorperhildung in Oesophagus, Inaug. Diss., Freiburg, 1887.

55. Miami, M.: Mioma dell'esofago, Gior. Ven. Sc. Med. **10:** 486, 1936.
56. Miller, J.: A Large Leiomyoma of the Esophagus, J. Path. & Bact. **17:** 278-281, 1912.
57. Milovanovich, M.: Ueber der Leiomyoma des Oesophagus und der Cardia, Wien. klin. Wchnschr. **27:** 753-755, 1914.
58. Moersch, H. J., and Harrington, S. W.: Benign Tumors of the Esophagus. Ann. Otol. Rhin. a. Larying. **53:** 800-817, 1944.
59. Monro, J.: Disputatio medica inauguralis de Dysphagia, Edimburg, 1797.
60. Oberhelman, H. A., Condon, J. B., and Guzauskas, A. C.: Leiomyoma of the Gastro Intestinal Tract, S. Clin. North American **32:** 111-122, 1952.
61. Ohsawa, T.: The Surgery of the Esophagus, Arch. Jap. Chir. **10:** 605-700, 1933.
62. Pape, R., and Spitznagel, K.: Ueber Oesophagus Myome, Fortschr. Röntgenstr. **44:** 616, 1931.
63. Patterson, E. J.: Benign Neoplasms of the Esophagus, Pennsylvania M. J. **36:** 244-246, 1933.
64. Pennes, A. E.: Leiomyosarcoma of the Esophagus, Am. J. Roentgenol. **48:** 336-340, 1942.
65. Pichler, R.: Ueber Einen Fall von Multiplen Leiomyomen des Oesophagus, Prager Med. Wchnschr. **22: 1897.**
66. Placitelli, G.: Su due casi di stenosi esofagea da tumori poco comuni, Boll. Sc. Med. **121:** 399-408, 1949.
67. Reher: Beitrag zur Kasuistik der Oesophagus Erkrankungen, Inaug. Diss., Leipzig, 1885.
68. Ribbert: Beitrage zur Kenntniss der Rabdomyome, Virchows Arch. **130:** 1882.
69. Rodino', di Miglione D.: Il leiomioma dell'esofago, Riv. chir. **7:** 117-122, 1941.
70. Rose, J. D.: Myomata of the Esophagus, Brit. J. Surg. **24:** 297-308, 1936.
71. Rossello, G.: I leiomioma dell'esofago, Arch. Chir. Tor. **6:** 133-181, 1950.
72. Samaia, N.: I tumori dell'esofago, Soc. Ed. Libraria, 197, 1917.
73. Sauerbruch, F., and O'Shaughnessy, L.: Thoracic Surgery. Baltimore, 1937, William Wood & Co., p. 333.
74. Schafer, P. W., and Kittle, C. F.: Esophageal Leiomyoma; Report of a Successful Resection, J. A. M. A. **133:** 1202-1205, 1947.
75. Schatzki, R., and Hawes, L. E.: The Roentgenological Appearance of Extramucosal Tumors of the Esophagus, Am. J. Roentgenol. **48:** 1-15, 1942.
76. Schnug, G. E.: Leiomyoma of the Cardioesophageal Junction, A. M. A. Arch. Surg. 65: 342-346, 1952.
77. Stewart, J. D.: Myoma of the Oesophagus With Associated Diverticula, A. M. A. Arch. Path. **12:** 77-84, 1931.
78. Sweet, R. H.: Leiomyoma of Esophagus (Case Records of Massachusetts General Hospital: Case 33252). New England J. Med. **236:** 955-957, 1947.
79. Sweet, R. H., Soutter, Lamar, and Valenzuela, Carlos: Muscle Wall Tumors of the Esophagus J. THORACIC SURG. **27:** 13-35, 1954.
80. Terracol, J.: Les maladies de l'esophage. (Tumeurs benignes de l'esophage), Masson 459-472, 1938.
81. Tonelli, L.: Studio sui tipi, le varianti istopathologiche, l'ordinamento dei tumori di natura muscolare, Arch. **5:** 295-620, 1942.
82. Tschlenow, S.: Ueber der Leiomyom des Oesophagus, Virchows Arch. path. Anat. 242: 1923.
83. Virchow, R.: Berlin, A. Hirschwald **3:** 128, 1863.
84. Winderen, L.: Godartede oesophagus tumores; betrivelse av two Kasus, Tidoskr. norske laegefor. **69:** 529-531, 1949.
85. Bigger, I. A., Kay, W. R., and Vinson, P. P.: Leiomyoma of the Esophagus. Report of a Case, Virginia M. Month **77:** 290-291, 1950.
86. Brizzolara, L. G., and Geraci, C. L.: Esophageal Leiomyoma, Surgery **35:** 465-469, 1954.
87. Flavell, G.: Leiomyoma of the Esophagus, Brit. J. Surg. **41:** 238-240, 1953.
88. Kenney, L. J.: Giant Intramural Leiomyoma of Esophagus, J. THORACIC SURG. **26:** 93-100, 1953.
89. Madden, W. C., and Olmstead, E. G.: Leiomyoma of the Esophagus: Report of a Case With Successful Resection, Ohio M. J. **46:** 974-975, 1950.
90. Myers, R. T., and Bradshaw, H. H.: Benign Intramural Tumors and Cysts of the Esophagus, J. THORACIC SURG. **21:** 470-482, 1951.
91. Nissen, R.: Bridging of Esophageal Defect by Pedicled Flap of Lung Tissue, Ann. Surg. **129:** 142-147, 1949.
92. Westerhorn, O.: Leiomyoma of the Esophagus, Acta chir. scandinav. **103:** 158, 1952.
93. Wilder, J. R., and Moore, S. W.: Leiomyoma of the Esophagus, A. M. A. Arch. Surg. 67: 595-602, 1953.
94. Zaslow, J., and Krasnoff, S. O.: Leiomyoma of the Esophagus (Report of a Case With Successful Enucleation of the Tumor), J. THORACIC SURG. **26:** 86-92, 1953.

INTRABRONCHIAL LIPOMA

A Case Report

Robert E. McCall, M.D., and William Harrison, M.D.
Kingsport, Tenn.

THE increasing availability of thoracic surgery, bronchoscopy, and their diagnostic adjuncts is reflected in the rising number of reported benign intrabronchial tumors. The lipoma ranks third in incidence among benign mesodermal intrabronchial tumors, but comprises a very small percentage of them.[15] Its rarity may be judged by the fact that we have been able to find only fifteen previously described surgically removed and histologically confirmed intrabronchial lipomas or fibrolipomas. Of these, nine were reported prior to 1950, whereas six have been reported since that time. The illustration of intrabronchial lipoma in Liebow's fascicle[15] is from an unreported case operated on in 1945, to the best of our knowledge.[16]

Rokitansky is credited in previous reports[8, 14] with describing the first intrabronchial lipoma, in 1854, discovered at necropsy. The first reported surgically removed lipoma was that of Kernan in 1927.[1] During the next twelve years three closely related tumors, fibrolipomas, were reported by Myerson in 1929,[2, 3] Wessler and Rabin in 1932,[5] and McGlade in 1939.[6] These tumors, along with others reported, are listed in Table I.

Table I. Reported Cases of Intrabronchial Lipoma, Surgically Treated

AUTHORS	YEAR	SEX	AGE	COLOR	LOCATION	TREATMENT
Kernan[1]	1927	M	50	W	Right main bronchus	Bronchoscopic excision
Myerson[2, 3]	1929	M	47	W	Left main bronchus	Bronchoscopic excision
Jackson & Jackson[4]	1932	F	64	W	Left main bronchus	Bronchoscopic excision
Wessler and Rabin[5]	1932	M	48		Left lower lobe	Bronchoscopic excision
McGlade[6]	1939	M	48	W	Left main bronchus	Bronchoscopic excision
Vinson and Pembleton[7]	1942	F	29	N	Left main bronchus	Bronchoscopic coagulation (pneumonectomy advised)
Watts, Clagett and McDonald[8]	1946	M	51	W	Left upper lobe bronchus	Left upper lobectomy
Whalen[9]	1947	M	61	W	Left upper lobe bronchus	Bronchoscopic excision
Lell[10]	1949	M	40	W	Right main bronchus	Bronchoscopic excision
Carlisle, Leary and McDonald[11]	1951	M	52	W	Left upper lobe bronchus	Left upper lobectomy
Som and Feuerstein[12]	1951	M	55	W	Right lower lobe bronchus	Bronchoscopic excision
		M	59	W	Right upper lobe bronchus	Bronchoscopic excision
Beaton and Heatly[13]	1952	M	36	N	Left main bronchus	Bronchoscopic excision
Smart[14]	1953	M	65	W	Trachea	Bronchoscopic excision
		M	60	W	Left main bronchus	Bronchoscopic excision

From Holston Valley Community Hospital, Kingsport, Tenn.
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The occurrence of these lipomas in three Negroes in a total of sixteen may be a chance incidence of no statistical significance, but, if such a high proportion continues as the number of reported cases increases, it will be an interesting contrast to the lower incidence of bronchogenic carcinoma in Negroes than white men. The general preponderance of men over women with pulmonary tumors exists in this series as well. More of these lipomas have been reported in the left bronchus than the right. It may be that this is due to the fact that they are all in the major bronchi, either stem bronchus or primary divisions where fat is normally found, and the left main bronchus exceeds the right in length, giving a greater chance of occurrence here. The lower reported incidence of lipomas in the trachea is difficult to reconcile, in view of the greater proportional length of this airway, and indicates the operation of factors other than chance in the development and location of these tumors.

The growth characteristics reported indicate that the intrabronchial lipoma is often pedunculated, occasionally sessile, and in one instance in dumbbell distribution that had a deep portion external to the cartilaginous plates.[11] Thus, in the last instance, the intrabronchial lipoma resembles the pattern seen in bronchial adenomas, and confirms by its continuity the hypothesis that these tumors arise from normal fat located external to the cartilage. The polypoid nature of the tumor is reflected in the frequency of bronchoscopic removal. Pulmonary resection was thought to be indicated because of distal suppurative disease in only three of the previously reported cases. It was actually performed in two. The lipoma, like other histologically benign tumors, may produce serious and prolonged illness, even death, when located in such a critically important position as a main bronchus.

The following case report demonstrates a common sequence of events seen in benign intrabronchial tumors, namely, partial then complete respiratory obstruction with distal suppuration and bronchiectasis. Hemoptysis, which occurred in this case, was mentioned in five of the previously reported cases.

CASE REPORT

This 60-year-old Negro man was first seen in the outpatient department on April 10, 1951, with the presenting signs and symptoms of pain in the left chest, cough, cyanosis, and dependent edema. During the preceding two or three months he had noted shortness of breath on exertion, orthopnea and had had several bouts of hemoptysis. His appetite remained good, and there had been no known weight loss. He was hospitalized one week later for study of possible carcinoma of the left lung.

The principal findings on examination at this time were: temperature 98.6° F., pulse 100, blood pressure 140/80, respirations 20. He appeared to be about the stated age, and moderately well nourished. There was dullness to percussion over the lower left chest with breath sounds diminished to absent over this area. Crepitant râles were present over the remainder of the left lung and the base of the right lung. The heart was enlarged to the left, as shown by percussed dullness and x-ray examination (Figs. 1 and 2). No peripheral edema or cyanosis was noted. The liver and spleen were not palpable. A blood count showed 4.5 million red blood cells, 13.5 Gm. hemoglobin, 12,800 white blood cells with 62 per cent neutrophils, 35 per cent lymphocytes, and 3 per cent eosinophils. Urinalysis was normal. Serologic test for syphilis was negative. Nonprotein nitrogen was 55 mg. per cent. Sputum smears and cultures were negative for acid-fast bacilli. An electrocardiogram showed left heart strain and digitalis effect.

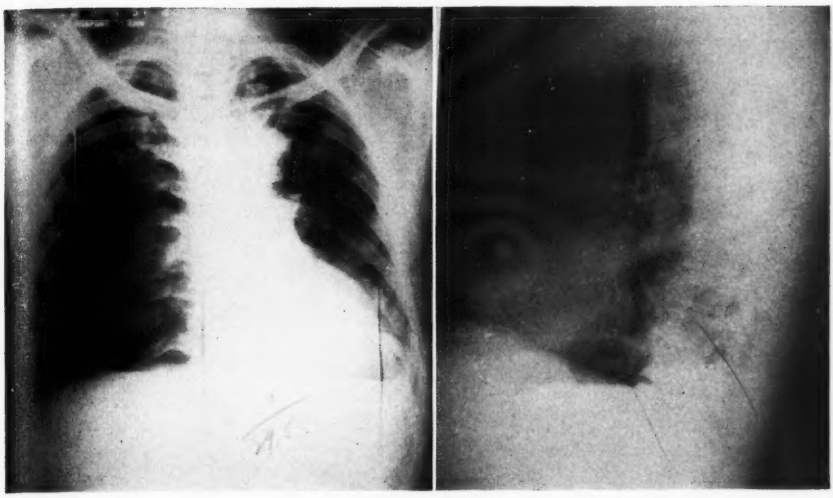

Fig. 1. Fig. 2.

Figs. 1 and 2.—Anteroposterior and lateral films of chest on first admission, April 26, 1951.

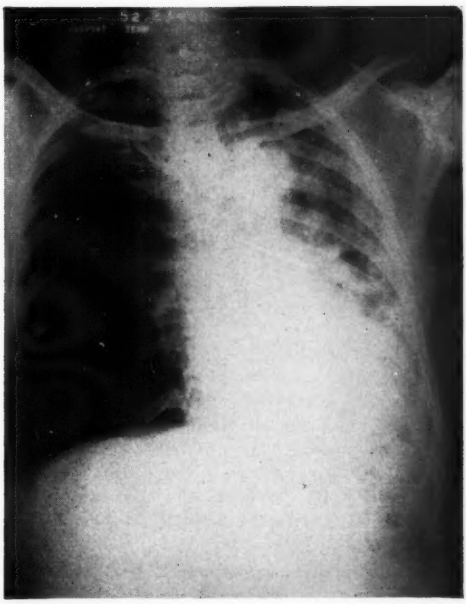

Fig. 3.—Film of chest on Sept. 2, 1952.

The patient was discharged from the hospital on May 1, 1951, with the diagnosis of cardiac failure and consolidation of the left lower lobe of the lung. Treatment with antibiotics, diuretics, digitalis, aminophylline, and laxatives had produced improvement. The possibility of a pulmonary neoplasm was not considered further at that time. A repeat x-ray of the chest, done ten days after discharge, showed infiltration of the left base and emphysema of the left upper lobe.

He was next seen in the outpatient department on Sept. 2, 1952, with the same symptoms, but some loss of weight and strength. The entire left side of the chest was dull to percussion with diminished to absent breath sounds. He was readmitted to the hospital on Sept. 9, 1952, when he was first seen by one of us (R. E. M.). The cardiac condition appeared clinically improved in that the chest:heart ratio was normal, there was no evidence of congestive failure, and the nonprotein nitrogen was 30 mg. per cent; however, the electrocardiogram still showed left heart strain and occasional ventricular premature beats. The increased consolidation of the entire left lung (Fig. 3) with mediastinal shift to the left but an improved cardiac status was noted. Other findings were essentially unchanged.

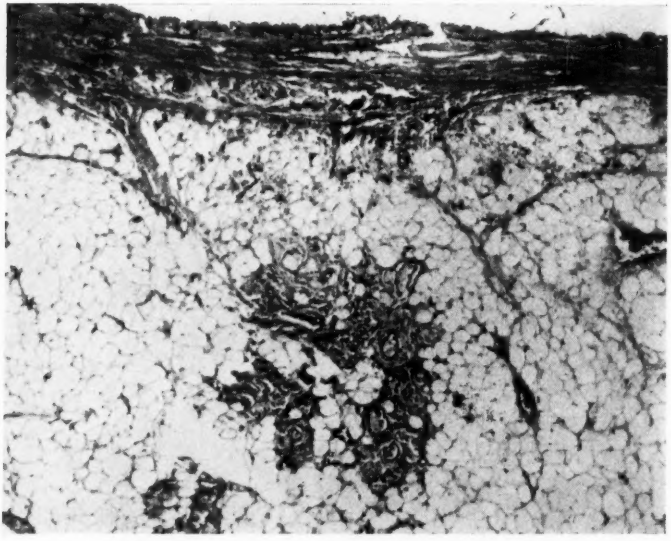

Fig. 4.—Microscopic appearance of bronchial lipoma showing respiratory epithelium and inclusion of mucous glands.

Bronchoscopic examination was carried out the next day, at which time a smooth globular tumor was found to block the left main bronchus almost completely just distal to the carina. A biopsy of the growth showed normal respiratory mucous membrane with some underlying fatty tissue. Following this, the productive cough stopped and remittent fever ranging from 102° to 103.6° F. developed. This was thought to be due to complete obstruction from edema associated with the biopsy. An hematocrit of 33 was raised by transfusions to 41 in preparation for operation.

A transpleural bronchotomy was done on Oct. 2, 1952. The tumor, which had a narrow pedicle, was easily removed. A pneumonectomy with transection of the bronchus distal to the bronchotomy site was performed because of the extensive suppurative disease of the lung.

The tumor measured 2.5 cm. in greatest dimension. It had a smooth multilobular surface, pale yellow color, and soft rubbery consistency. On cut section it was solid with a pale lemon-yellow, slightly translucent appearance. Small lobular areas were partially surrounded by thin white septa. Microscopic examination showed it to be composed of mature adipose tissue with the inclusion of many mucous glands (Figs. 4 and 5). The epithelium was denuded in some areas but, where present, was of respiratory type with areas of squamous metaplasia.

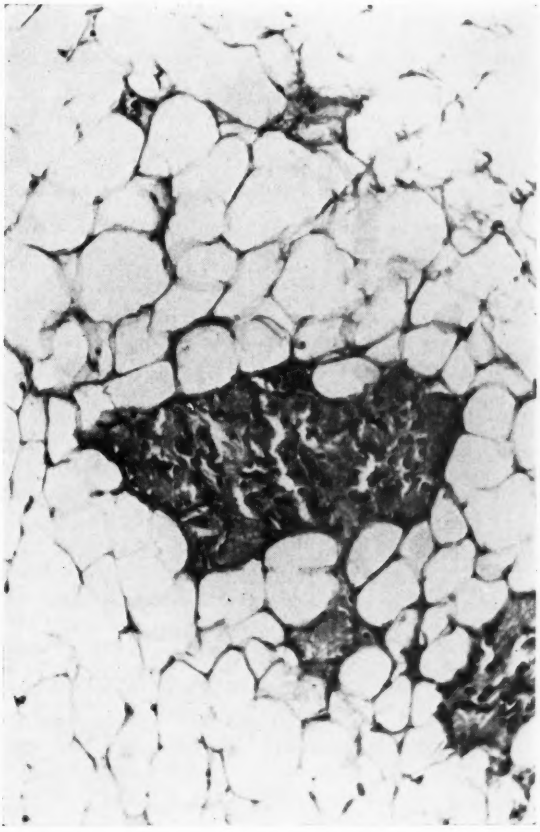

Fig. 5.—Microscopic appearance of lipoma showing mucous glands surrounded by adipose tissue.

The left lung, submitted separately, weighed 800 grams and was the seat of considerable bronchiectasis and fibrosis involving both lobes, with extensive acute and organizing focal pneumonitis.

During the early follow-up period the patient remained well, free of cough, and gained in strength and weight. He occasionally lapsed into congestive failure because he did not follow prescribed treatment, but always responded well to hospitalization. He was most recently seen in congestive failure in March, 1954. On bed rest, diuretics, and redigitalization he lost 30 pounds of edema. The x-ray appearance of the left thorax has been stable with no change in the level of the diaphragm or position of the heart and trachea. He left the hospital compensated at rest.

REFERENCES

1. Kernan, J. D.: Three Unusual Endoscopic Cases, Laryngoscope **37**: 62-64, 1927.
2. Myerson, M. C.: Benign Neoplasms of the Bronchus: Report of a Case of Fibrolipoma of the Left Main Bronchus Removed Through Bronchoscope, Am. J. M. Sc. **176**: 720-726, 1928.
3. Myerson, M. C.: Benign Neoplasms of the Bronchus: Report of a Case of Bronchoscopic Removal of Fibrolipoma From the Left Main Bronchus, Arch. Otolaryng. **9**: 376-385, 1929.
4. Jackson, C., and Jackson, C. L.: Benign Tumors of the Trachea and Bronchi: With Especial Reference to Tumor-Like Formations of Inflammatory Origin, J. A. M. A. **99**: 1747-1754, 1932.
5. Wessler, H., and Rabin, C. B.: Benign Tumors of the Bronchus, Am. J. M. Sc. **183**: 164-180, 1932.
6. McGlade, T. H.: Fibro-lipoma of the Bronchus: Report of a Case, Ann. Otol., Rhin. & Laryng. **48**: 240-243, 1939.
7. Vinson, P. P., and Pembleton, W. E.: Lipoma of the Left Main Bronchus: Report of a Case and Review of Literature, Arch. Otolaryng. **35**: 868-870, 1942.
8. Watts, C. F., Clagett, O. T., and McDonald, J. R.: Lipoma of the Bronchus: Discussion of Benign Neoplasms and Report of a Case of Endobronchial Lipoma, J. THORACIC SURG. **15**: 132-143, 1946.
9. Whalen, E. J.: Lipoma of the Bronchus, Ann. Otol., Rhin. & Laryng. **56**: 811-818, 1947.
10. Lell, W. A.: Report of a Case of Fibrolipoma of Right Main Bronchus: Bronchoscopic Removal, Ann. Otol., Rhin. & Laryng. **58**: 1124-1134, 1949.
11. Carlisle, J. C., Leary, W. V., and McDonald, J. R.: Endobronchial Lipoma: Report of Case, Proc. Staff Meet., Mayo Clin. **26**: 103-106, 1951.
12. Som, M. L., and Feuerstein, S. S.: Endoscopic Removal of Lipoma of the Bronchus: Report of 2 Cases, A. M. A. Arch. Otolaryng. **54**: 341-346, 1951.
13. Beaton, A. H., and Heatly, C. A.: Fat in the Tracheo-bronchial Tree With Report of a Case of True Lipoma of the Bronchus, Ann. Otol., Rhin. & Laryng. **61**: 1-9, 1952.
14. Smart, J.: Intrathoracic and Intrabronchial Lipomata, Brit. J. Tuberc. **47**: 1-6, 1953.
15. Liebow, A. A.: Tumors of the Lower Respiratory Tract, Atlas of Tumor Pathology, Armed Forces Institute of Pathology, Sec. V, Fascicle 17, pp. 119, 128-129, 1952.
16. DeCoursey, E.: Personal communication.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-FIFTH ANNUAL MEETING

APRIL 24, 25, AND 26, 1955

CHALFONTE-HADDON HALL, ATLANTIC CITY, N. J.

PROGRAM

Sunday Morning, April 24, 1955

8:30 A.M. Business Meeting.

9:00 A.M. Scientific Session: REGULAR PROGRAM.

1. **Pleurobiliary and Bronchobiliary Fistulae.** Herbert D. Adams, Boston, Mass.
2. **Staphylococcal Pneumonia in Infants: A Report of Ten Cases.** W. E. Bloomer (by invitation), R. E. Cooke (by invitation), G. E. Lindskog, and S. Giammona (by invitation), New Haven, Conn.
3. **Management of Massive Hemoptysis, Not Due to Pulmonary Tuberculosis or Neoplasm.** J. L. Ehrenhaft and Rodman E. Taber (by invitation), Iowa City, Iowa.
4. **The Significance of the Anterior Segment in Bronchiectasis.** Richard H. Overholt and Wilford B. Neptune (by invitation), Boston, Mass.
5. **"Spontaneous" Rupture of the Esophagus.** George W. B. Starkey (by invitation), Boston, Mass.
6. **One-Stage Pharyngo-esophageal Diverticulectomy.** J. D. Mortensen (by invitation), O. Theron Clagett, Herbert W. Schmidt, and Howard K. Gray, Rochester, Minn.

Sunday Afternoon, April 24, 1955

2:00 P.M. Scientific Session: REGULAR PROGRAM.

7. **The Diagnosis of Acquired Valvular Disease by Left Heart Pressure Recordings.** Don. L. Fisher (by invitation), Pittsburgh, Pa.
8. **A Laboratory and Clinical Evaluation of Operations for Coronary Artery Disease.** David S. Leighninger (by invitation), Cleveland, Ohio.
9. **Technical Factors Which Favor Mammary-Coronary Anastomosis With Reports of Forty Cases of Human Coronary Artery Disease Thus Treated.** Arthur Vineberg and William Buller (by invitation), Montreal, Que.
10. **Analysis of 50 Patients Treated by Mitral Commissurotomy Five or More Years Ago.** Robert P. Glover, Thomas J. E. O'Neill, and O. Henry Janton (by invitation), Philadelphia, Pa.
11. **Evaluation of Techniques for the Repair of Auricular and Ventricular Septal Defects.** Earle B. Kay, Frederick S. Cross (by invitation), and Henry A. Zimmerman (by invitation), Cleveland, Ohio.
12. **Experiences With Surgical Repair of Atrial Septal Defects.** Elton Watkins, Jr., and Robert E. Gross, Boston, Mass.

Monday Morning, April 25, 1955

8:30 A.M. Scientific Session: THORACIC SURGICAL FORUM.

13. **Anastomosis of Right Auricle to Pulmonary Artery in the Treatment of Tricuspid Atresia.** Elliott S. Hurwitt, Dennison Young (by invitation), and Doris J. W. Escher (by invitation), New York, N. Y.

14. **Autogenous Thoracic Aorta Grafts of Pericardium and Nylon Net Using a Thrombin-Fibrinogen Coagulum.** Richard H. Adler (by invitation), Denver, Colo.

15. **The Lymphatic Drainage of Silver-Coated Radioactive Gold Colloids Following Intra-thoracic Administration to Pneumonectomized Dogs.** P. F. Hahn (by invitation), Robert A. Matuska (by invitation), Robert I. Carlson, Stewart H. Auerbach (by invitation), and George R. Meneely (by invitation), Nashville, Tenn.

16. **Circumferential Suture of the Mitral Ring—A Possible Method for the Surgical Correction of Mitral Insufficiency.** Julio C. Davila, Robert G. Trout, Bart S. Iaia, and Frank Mansure (all by invitation). Sponsored by Robert P. Glover, Philadelphia, Pa.

17. **Studies Upon the Physiological Function of the Human Vagus Nerve in Various Pathological Pulmonary States.** Osler A. Abbott, W. E. VanFleit (by invitation), E. R. Duschesne (by invitation), and A. E. Roberto (by invitation), Emory University, Ga.

18. **Temporary Unilateral Occlusion of the Pulmonary Artery in the Preoperative Evaluation of Thoracic Patients. A Preliminary Report.** Herbert Sloan and Joe D. Morris (by invitation), Ann Arbor, Mich.

19. **Coronary Perfusion for Longer Periods of Cardiac Occlusion Under Hypothermia.** Marvin L. Gliedman (by invitation) and Norman E. Shumway (by invitation). Sponsored by F. John Lewis, Minneapolis, Minn.

20. **Blood Volume Studies in Thoracic Surgical Procedures Using Radioactive Iodinated Human Serum Albumin (RIHSA).** Clifford F. Storey, Charles G. Foster (by invitation), and Thomas G. Mitchell (by invitation), St. Albans, N. Y.

21. **Induced Cardiac Arrest for Intracardiac Surgical Procedures.** Conrad R. Lam, Thomas Geoghegan (by invitation), and Alfredo Lepore (by invitation), Detroit, Mich.

22. **Open Left Heart Surgery in Dogs During Cardiac Arrest Below 10° C. Temperature With and Without Extracorporeal Circulation.** Frank Gollan, Reginald Phillips, Jr., James T. Grace, and Raymond M. Jones (all by invitation), Nashville, Tenn.

23. **Physiological Observations in Experimental Pulmonary Insufficiency.** Robert G. Ellison, Walter J. Brown, Jr. (by invitation), Elmer E. Hague, Jr. (by invitation), and William F. Hamilton (by invitation), Augusta, Ga.

24. **Experimental Transposition of the Great Vessels.** Sanford E. Leeds, Morris M. Culliner (by invitation), and Sherman H. Strauss (by invitation), San Francisco, Calif.

25. **Controlled Total Body Arterial Perfusion for Open Intracardiac Surgery.** Herbert E. Warden (by invitation), Richard A. DeWall (by invitation), J. Bradley Aust (by invitation), Morley Cohen (by invitation), Raymond C. Read (by invitation), and C. Walton Lillehei, Minneapolis, Minn.

26. **The Arterialization of Blood as It Applies to the Mechanical Heart-Lung Apparatus.** F. D. Dodrill and Alfred Lui (by invitation), Detroit, Mich.

27. **The Alveolar Carbon Dioxide Tension During Intrathoracic Operations.** Thomas F. Nealon (by invitation), George J. Haupt (by invitation), and John H. Gibbon, Jr., Philadelphia, Pa.

Monday Afternoon, April 25, 1955

2:00 P.M. Executive Session. (Limited to Active and Senior Members.)

3:00 P.M. Scientific Session: REGULAR PROGRAM.

Remarks by the President, Edward S. Welles, Saranac Lake, N. Y.

28. **Lower Esophageal Web.** Walter F. Bugden and J. Ernest Delmonico (by invitation), Syracuse, N. Y.

29. **Lymphoma of the Lung and Pleura.** Willard Van Hazel and Robert J. Jensik (by invitation), Chicago, Ill.

30. **Chest Wall Prosthesis.** Bert H. Cotton, George A. Paulsen (by invitation), and Jack Dykes (by invitation), Los Angeles, Calif.

31. **Thymic Neoplasms.** Donald B. Effler and Lawrence J. McCormack (by invitation), Cleveland, Ohio.

6:30 P.M. Cocktail Party, Chalfonte-Haddon Hall. Informal.

Tuesday Morning, April 26, 1955

9:00 A.M. Scientific Session: REGULAR PROGRAM.

32. **Pulmonary Resection in Active Cavitary (Open-Positive) Tuberculosis.** Robert H. Holland (by invitation), John W. Bell (by invitation), and Edward S. Welles, Sunmount, N. Y.

33. **Simultaneous Bilateral Resection for Pulmonary Tuberculosis in Mental Patients.** F. John Lewis, M. Taufic (by invitation), B. Zimmerman (by invitation), and J. F. Perry (by invitation), Minneapolis, Minn.

34. **Blood Volume Studies in Pulmonary Tuberculosis.** John H. Kehne (by invitation) and Felix A. Hughes, Memphis, Tenn.

35. **Surgical Treatment in Tuberculosis Complicated With Pulmonary Emphysema.** Robert W. Newman, Perry M. Huggin, Charles L. Butler, and Medford C. Bowman (all by invitation), Knoxville, Tenn.

36. **Surgery for Cavitary Tuberculosis in Patients With a Single Lung.** Francis M. Woods and Norman J. Wilson, Boston, Mass.

37. **Surgical Treatment of Pulmonary Histoplasmosis With MRD-112 as an Adjunct.** John W. Polk (by invitation), Chas. A. Brasher (by invitation), João de Castro (by invitation), and W. W. Buckingham, Kansas City, Mo.

Tuesday Afternoon, April 26, 1955

2:00 P.M. Scientific Session: REGULAR PROGRAM.

38. **The Development of the Pleural Partition to Prevent Overexpansion of the Lung Following Partial Lung Resection.** Lyman A. Brewer, III, Angel F. Bai (by invitation), and W. M. Jones (by invitation), Los Angeles, Calif.

39. **Operative Thoracic Ductography: Report of Three Cases Illustrating Its Value.** Allan Stranahan, Ralph D. Alley, and Harvey W. Kausel (by invitation), Albany, N. Y.

40. **Complications of Mitral Valve Surgery With Particular Reference to the Post-Commissurotomy Syndrome.** Charles B. Ripstein, New York, N. Y.

41. **The Surgical Treatment of Aortic Stenosis.** C. P. Bailey, H. E. Bolton (by invitation), W. L. Jamison (by invitation), and H. T. Nichols (by invitation), Philadelphia, Pa.

42. **The Use of Shunts in the Resection of the Thoracic Aorta.** J. Maxwell Chamberlain, Robert Klopstock, Peter Parnassa (by invitation), and A. Grant (by invitation), New York, N. Y.

43. **Patent Ductus Arteriosus With Hypertension: Analysis of Cases in Which Operation Was Performed.** F. Henry Ellis (by invitation), John W. Kirklin, and Earl H. Wood (by invitation), Rochester, Minn.

Notice

Those who are on the program are reminded that by action of the Council all papers are to be handed to the Recorder *immediately* after their presentation. Any paper which is not thus submitted immediately runs a risk of not being published.